MEDICAL PROCEEDINGS

MEDIESE BYDRAES

A South African Journal for the Advancement of Medical Science

'n Suid-Afrikaanse Tydskrif vir die Bevordering van die Geneeskunde

P.O. Box 1010 · Johannesburg Posbus 1010 · Johannesburg

Editor : Redakteur

H. A. Shapiro, B.A., Ph.D., M.B., Ch.B., F.R.S.S.Af.

Vol. 7 21 October 1961 Oktober 21 No. 21

EDITORIAL · REDAKSIONEEL

CARCINOMA OF THE UTERUS

The recently issued Twelfth Volume of the *Annual Report on the Results of Treatment in Carcinoma of the Uterus** has been issued by Radiumhemmet, Stockholm, under the patronage of the International Federation of Gynecology and Obstetrics. This Report covers a statement of the results obtained during the period 1945 to 1954, inclusive. Because opinion still differs on what lesion Stage O actually represents, all Stage O cases were excluded from the present Report, although the Editorial Committee appreciated the importance of the diagnosis of the Stage O intraepithelial lesion and the necessity of separating such cases from those of invasive carcinoma.

A word of warning is sounded against drawing too far-reaching conclusions from the data presented by the various collaborating institutions in respect of the treatment given.

'Differences in the therapeutic results may be due to many factors besides the method of treatment used, for instance the proportion of patients lost sight of, and the quality of the material, whether intrinsic or connected with voluntary or involuntary selection. Another fact that should be considered is that random fluctuations may lead to differences in the results. Even differences as high as 12% in the recovery rates between two samples of 100 cases each (and where the chance of recovery lies around 50-50) occur fairly often by pure random, in fact as often as in one sample out of 10' (p. 327).

* *Annual Report on the Results of Treatment in Carcinoma of the Uterus. Twelfth Volume: Statements of Results Obtained in* 1945 *to* 1954, *Inclusive.* Published under the Patronage of the International Federation of Gynecology and Obstetrics. *Editor*: Dr. H. L. Kottmeier. *Editorial Office*: Stockholm 60, Sweden.

KARSINOOM VAN DIE BAARMOEDER

Die onlangs gepubliseerde Twaalfde Deel van die *Annual Report on the Results of Treatment in Carcinoma of the Uterus** is deur Radiumhemmet, van Stockholm, onder beskerming van die Internasionale Federasie vir Ginekologie en Verloskunde uitgegee. Hierdie verslag bevat 'n uiteensetting van die resultate wat gedurende die tydperk 1945 tot 1954, albei jare inbegrepe, behaal is. Omdat daar nog steeds meningsverskil bestaan oor die letsel wat in werklikheid deur Stadium O verteenwoordig word, is geen Stadium O-gevalle by die huidige verslag ingesluit nie, hoewel die Redaksionele Komitee wel deeglik bewus is van die belangrikheid van die diagnose van die Stadium O-binne-epiteelletsel en die noodsaaklikheid om hierdie gevalle van invasie-karsinoom te skei.

'n Woord van waarskuwing moet geuit word teen die neiging om té verregaande gevolgtrekkings te maak aan die hand van die gegewens wat deur die verskillende samewerkende inrigtings ten opsigte van behandelingsmetodes voorgelê is.

'Verskille in die terapeutiese resultate kan die gevolg wees van talle faktore behalwe die behandelingsmetode wat toegepas is—byvoorbeeld, die proporsie pasiënte wat uit die oog verloor is, en die kwaliteit van die materiaal, hetsy intrinsiek of voortspruitende uit willekeurige of onwillekeurige seleksie. 'n Ander feit wat in gedagte gehou moet word, is

* *Annual Report on the Results of Treatment in Carcinoma of the Uterus. Twelfth Volume: Statement of Results Obtained in* 1945 *to* 1954 *Inclusive.* Gepubliseer onder die beskerming van die Internasionale Federasie vir Ginekologie en Verloskunde. Redakteur: Dr. H. L. Kottmeier. Redaksionele Kantoor: Stockholm 60, Swede.

CARCINOMA OF THE CERVIX

The Report concludes that the present *International Classification of the Stages of Carcinoma of the Uterine Cervix* does not provide sufficient data on the composition of the material. The criteria for allotting cases to the appropriate stage have been shown, by experience, to be insufficiently well defined. The Editorial Committee therefore proposes to raise the question of a revision of the present system of international staging, without changing the principles of the present rules for staging but with the object of making the definitions more precise, so as to make the comparison of the results of treatment more reliable.

The relative apparent 5-year recovery rate calculated for each of the 4 stages for the 10-year period 1945-1954 is shown in Table 1.

TABLE 1

	No. of Patients Treated	*Alive with no Evidence of the Disease*	*Relative Apparent Recovery Rate*
Stage I	20,267	14,398	71.0
Stage II	34,672	17,066	49.2
Stage III	29,562	7,716	26.1
Stage IV	6,334	450	7.1
Unclassified ...	5	2	
	90,840	39,632	43.6

CARCINOMA OF THE CORPUS

In considering the definitions of the different varieties of endometrial carcinoma, the Editorial Committee found the classification recommended to be unsatisfactory and to have caused much misunderstanding. A revision of the classification and the staging of carcinoma of the body of the uterus is therefore under consideration.

The relative apparent 5-year recovery rate calculated for the various stages and groups is shown in Table 2.

TABLE 2

	No. of Patients Treated	*Alive with no Evidence of the Disease*	*Relative Apparent Recovery Rate*
Stage I, group 1	9,517	6,957	73.1
Stage I, group 2	4,911	2,234	45.5
Stage II	2,237	488	21.8
	16,665	9,679	58.1

dat toevallige wisselinge aanleiding tot verskille in die resultate kan gee. Selfs verskille van soveel soos 12% in die herstelsyfers tussen twee monsters van 100 gevalle elk (en waar die kanse op herstel in die omgewing van 50-50 is) kom betreklik dikwels per blote toeval voor, trouens so dikwels soos by een monster uit 10' (bl. 327).

KARSINOOM VAN DIE SERVIKS

Die verslag raak tot die gevolgtrekking dat die huidige *Internasionale Klassifikasie van die Stadiums van Karsinoom van die Baarmoedernek* nie voldoende gegewens oor die samestelling van die materiaal verstrek nie. Die ondervinding het geleer dat die maatstawwe vir die indeling van gevalle in geskikte stadiums nie goed genoeg omskryf is nie. Die Redaksionele Komitee is derhalwe van plan om die kwessie van 'n hersiening van die huidige stelsel van internasionale indeling in stadiums te berde te bring sonder enige verandering in die beginsels van die huidige reëls vir stadiumindeling, maar met die doel om die omskrywings presieser te maak sodat die vergelyking van behandelingsresultate betroubaarder kan wees.

Die betrokke skynbare 5-jaar-herstelsyfer soos bereken vir elk van die 4 stadiums gedurende die 10 jaar vanaf 1945 tot 1954 word aangetoon in Tabel 1.

TABEL 1

	Aantal pasiënte behandel	*Nog-lewend met geen tekens van die siekte*	*Relatiewe skynbare herstelpersentasie*
Stadium I	20,267	14,398	71.0
Stadium II ...	34,672	17,066	49.2
Stadium III ...	29,562	7,716	26.1
Stadium IV ...	6,334	450	7.1
Ongeklassifiseer	5	2	
	90,840	39,632	43.6

KARSINOOM VAN DIE CORPUS

By die oorweging van die omskrywings van die verskillende soorte endometriumkarsinome, het die Redaksionele Komitee bevind dat die aanbevole klassifikasie onbevredigend was en dat dit aanleiding tot heelwat misverstand gegee het. 'n Hersiening van die klassifikasie en stadiumindeling van karsinoom van die liggaam van die baarmoeder word derhalwe oorweeg.

Die relatiewe skynbare 5-jaar-herstelsyfer soos bereken vir die verskillende stadiums en groepe word in Tabel 2 aangetoon.

Die globale relatiewe skynbare 5-jaar-herstelsyfer van 58.1% in die huidige deel is die hoogste wat tot dusver in hierdie *Annual Reports* aangekondig is.

The over-all relative apparent 5-year recovery rate of 58.1% noted in the present volume is the highest yet presented in these *Annual Reports*, the lowest figure having been recorded in Vol. 8 (1946), when the recovery rate was 52.4%.

STABILITY OF RECOVERY IN CARCINOMA OF THE CERVIX AND CARCINOMA OF THE BODY

For this comparison all the cases reported in these 2 categories and treated over the period 1930-1954, were analysed.

Deaths due to intercurrent disease occurred more frequently among the corpus than among the cervix cases, because of the higher average age of the corpus patients.

It is also interesting that, excluding death due to intercurrent disease, the chance for a patient who has survived 5 years, to survive 5 more years, is 71.3% for carcinoma of the cervix and 75.6% for carcinoma of the corpus. This is contrary to the view held by many investigators that the risk for a late recurrence is relatively very much greater in corpus than in cervix cases. The tendency shown by the corpus patients towards a higher stability of the curability between 5 and 10 years after treatment appears to be well substantiated.

In future editions of these *Annual Reports* an attempt will be made to include other types of malignant tumours in the female pelvis. This will undoubtedly be an addition of the greatest value and will enhance the usefulness of this international analysis of the treatment of carcinoma occurring in the female pelvis.

Die laagste syfer het verskyn in Deel 8 (1946), toe die herstelsyfer 52.4% was.

TABEL 2

	Aantal pasiënte behandel	*Nog-lewend met geen tekens van die siekte nie*	*Relatiewe skynbare herstelpersentasie*
Stadium I, groep 1	9,517	6,957	73.1
Stadium I, groep 2	4,911	2,234	45.5
Stadium II	2,237	488	21.8
	16,665	9,679	58.1

STABILITEIT VAN HERSTEL VAN KARSINOOM VAN DIE SERVIKS EN KARSINOOM VAN DIE CORPUS

Vir hierdie vergelyking is al die gevalle wat in hierdie 2 kategorieë gerapporteer en gedurende die tydperk 1930-54 behandel is, ontleed.

Sterfgevalle ten gevolge van tussenkomende siektes het meer dikwels onder die corpus- as onder die serviks-gevalle voorgekom weens die hoër gemiddelde ouderdom van corpus-pasiënte.

Dit is ook interessant dat, met uitsondering van sterfgevalle ten gevolge van tussenkomende siektes, die kanse van 'n pasiënte wat die kwaal reeds 5 jaar lank oorleef het om 'n verdere 5 jaar te lewe 71.3% in die geval van karsinoom van die serviks en 75.6% in die geval van karsinoom van die corpus is. Dit is in stryd met die sienswyses van talle ondersoekers, naamlik dat die gevaar van 'n laat herverskyning van die siekte betreklikerwyse veel groter in corpus- as in serviksgevalle is. Die neiging van corpus-pasiënte tot groter stabiliteit van geneesbaarheid tussen 5 en 10 jaar ná behandeling skyn goed gegrond te wees.

In toekomstige uitgawes van dié *Annual Reports* sal 'n poging aangewend word om ook ander soorte kwaadaardige tumors van die vroulike bekken in te sluit. Dit sal ongetwyfeld 'n byvoegsel van die allergrootste waarde wees, en sal hierdie internasionale ontleding van die behandeling van karsinoom van die vroulike bekken nog nuttiger maak.

ABSTRACT

THE IMPORTANCE OF HYSTEROSALPINGOGRAPHY IN GENITAL TUBERCULOSIS

Clinical observations relating to 40 patients, in whom bacteriological and histological examinations, as well as hysterosalpingography, were carried out prior to operations, have shown that hysterosalpingography is of great importance in the field of genital tuberculosis. Genital tuberculosis may be present even when bacteriological tests repeatedly yield negative results. Histological examination of material obtained by abrasion usually reveals signs of a chronic, non-specific mucosal inflammation or of so-called pseudo-tuberculous changes. If, in these cases where the diagnosis is difficult to establish, hysterosalpingography provides a picture characteristic of tuberculosis, it may be considered as certain that genital tuberculosis is in fact present. Where the histological examination of material obtained by abrasion yields negative results, this simply means that the uterine mucosa has healed, while negative results in the bacteriological examinations only indicate that the adnexal process has healed. The disease as such may continue to exist.

Apart from its diagnostic use, hysterosalpingography is important in another respect, in that it makes it possible to record the peristaltic movements of the Fallopian tube; such recordings may assist the physician to decide whether an operation is indicated and what surgical procedure should be employed. Hysterosalpingography does not replace bacteriological and histological examinations, nor is the converse true; on the contrary, the three methods complement each other.

[Kardos, F. (1959): Zschr. Geburtsh., **153**, 137].

ACCESSORY BREAST TISSUE

A. LEE MCGREGOR, F.R.C.S., ENG., CH.M., EDIN.
Johannesburg

Accessory breast tissue is a neglected subject. The *British Journal of Surgery* was first published in 1913. Up to the end of 1960, this journal has contained but 3 short reports each dealing with a single case of accessory breast tissue.

In 1927 Purves and Hadley[38] recorded a case of aberrant breast tissue in each labium majus, stating this to be the first case of the kind recorded in England. In 1936 Noronha[37] reported the case of a woman with cystic degeneration in aberrant breast tissue in each axilla which he called supernumerary breasts. Andreasen[3] recorded a case with carcinoma in aberrant axillary tissue.

There is no reference to aberrant breast tissue in the index of Raven's 6-volume work on cancer,[40] nor is the subject mentioned in Ackerman's *Surgical Pathology*.[1]

Haagensen[28] makes brief reference to supernumerary breasts and nipples but none to aberrant breast tissue.

As far as I can determine, accessory breast tissue is not mentioned in the *South African Medical Journal* from 1927 up to the end of 1960.

The objects of this paper are to report the incidence of accessory breast tissue, to discuss its pathological significance, to review the literature, and to report 11 additional cases. Polythelia *per se* is not dealt with.

The embryonal milk line of Schultz extends from the axilla to the groin and the labium majus. Accessory breast tissue may occur at any situation on this line or it may be found off the line, e.g. face, ear, neck, arm, thigh, buttock, scapula, sternum, etc.

No explanation is given for these bizarre occurrences, which are referred to as freaks of nature. However, as de Cholnoky[12] points out, the opossum has a breast in the midline of the thorax, another in the midline of the abdomen in addition to numerous other breasts. In the Cetacea all breast tissue disappears except for a breast in each vulva. In Nutria (Myocastor) the breasts are behind the scapula, and in *Lagostomus maximus* and some other rodents, the breasts occur on the dorsolateral aspects of the thighs. In 1836 Geoffroy Saint-Hilaire surmised that the line of human descent was via an ancestor with multiple breasts and, in 1871, in the *Descent of Man*, Charles Darwin considered accessory breasts to represent a reversion to an ancestral type.

Whereas accessory breast tissue off the milk line is rare, it is common along the line of mammary genesis. Bresslau[7] reported, amongst Japanese, that accessory nipples occurred in 1.5% of males and 5% of females, whereas Guest[27] found only 80 cases in 20,000 English children, suggesting a racial factor.

de Cholnoky[13] found the incidence of accessory axillary breast tissue to be 6% in 2,000 female hospital cases. Fenech[20] in England reported only 7 instances of aberrant breast tissue in 600,000 hospital admissions. Deaver and McFarland[11] found that over 10,000 cases of polymastia were recorded in the literature prior to 1917.

THE SIGNIFICANCE OF ACCESSORY BREAST TISSUE

Geschickter's opinion[22] is that congenital anomalies of the breast are usually of academic rather than of therapeutic interest. Cole[10] states that tumours of aberrant breast tissue are of much more importance than those of accessory breast tissue, chiefly because of the frequency of cancer in the former. de Cholnoky[12] states that aberrant breast tissue is more liable to tumours, etc. than other varieties of accessory breast tissue. Matti[36], reviewing the evidence, states that axillary aberrant mammary tissue, but not that of supernumerary breasts, is more prone to malignant changes than is the normal breast. Masor[34] agrees with Andreasen,[3] and Christopher,[9] that aberrant breast tissue is particularly liable to tumour formation.

In summary, then, supernumerary breasts are not more liable than normal ones to the occurrence of cancer, whereas aberrant breast tissue is prone to this development.

CLASSIFICATION

There is confusion in the usage of terms descriptive of breast anomalies. Some authors[11, 22, 43] use the term *polymastia* as an all-embracing one for all types of congenital breast anomalies. Accessory breast tissue has the same implication. Others[10, 19, 34] use the term *aberrant* to describe mammary tissue which has neither

nipple nor areola and may therefore occur on or off the milk line. Thus the term *aberrant*, as applied to the breast, is used in a different sense from that in its application to the testis or thyroid.

The following classification of congenital accessory mammary tissue is suggested:

ACCESSORY MAMMARY TISSUE

1. Supernumerary:
   (*a*) Nipples.
   (*b*) Breasts with nipples.
2. Aberrant:
   Breast tissue devoid of nipples or areola.

These terms are applicable whether the anomalous tissue is on or off the milk line.

Because of the increased liability of aberrant breast tissue to pathological changes, more particularly carcinoma, it is of special interest to the general practitioner and the surgeon.

ABERRANT BREAST TISSUE

Such anomalies are commonly first noted in pregnancy, though they may be seen at puberty. They usually occur in proximity to the normal breast, most often in the axilla, but they may be found below the collar bone, on the sternum, in the labium majus, etc.

Falk[19] reports a case of carcinoma in a nodule of mammary tissue in the mid-sternal line. He reviews the literature up to 1950. His analysis is as follows:

Razemon and Bizard[41] collected all the cases of aberrant mammary tumours up to 1929. They totalled 76, of which 4 were malignant: 28 axillary; 10 sternal; and 5 subclavicular. Only one occurred in a male. Giacobbe[23] recorded one case of carcinoma in aberrant axillary mammary tissue. In 1932, however, Biancheri[4] reported what he assessed to be only the thirty-first case of cancer in aberrant axillary tissue. Massabuau and Gilbert[35] encountered 3 aberrant mammary cancers within a few months, 1 axillary, 1 subclavicular and 1 juxta-sternal. Erdman[18] reported 1,054 breast operations of which 573 were for cancer. In this series there were 7 cases of aberrant mammary tumours of which 3 were malignant.

Ravdin[39] recorded a carcinoma in subclavicular mammary tissue. Schmidt and Tannwald[47] found a large subclavicular aberrant mammary collection. de Cholnoky[12] recorded 40 cases of aberrant breast tissue in the axilla, all in females. Eleven operations were done and no malignancy found. Francheschini[21] reported a case in a woman of 64 who had noticed a subclavicular nodule since childhood. It enlarged with each pregnancy. It grew suddenly at the age of 63 and radical operation for cancer was carried out. Geschickter[22] records 7 cases of cancer in aberrant breast tissue, all of which occurred in the axilla. He stresses the great malignancy of the condition.

From 1950 to 1960 there have appeared in the world literature 29 articles dealing with accessory breast tissue. Siegler and Gordon[49] found 10 cases of supernumerary breasts in the vulva reported up to 1947. They point out that in the Cetacea all breast tissue normally disappears except for one mamma in each vulva. They state, furthermore, that the anatomic situation and the histology of supposedly aberrant mammary tissue in this situation make it wellnigh impossible to distinguish it from vestiges of Wolffian or mesonephric duct origin. Fibroadenoma occurred in 3 of the cases and adenocarcinoma in one.

Several other cases of vulval breast tissue are reported to the end of 1960: Della Romana de Compte and Radice,[14] Looney *et al.*[32] and Slepykh,[50] each reported a case of accessory vulval mammary gland. Dubrausky[17] described a pedunculated cystic breast growing from a mammary rudiment in the right labium majus. Hendrix and Behrman[29] reported another case of adenocarcinoma arising in a supernumerary mammary gland in the vulva. Gomes[25] devotes attention to the occurrence of supernumerary breasts and nipples in the male.

There are 11 papers dealing with supernumerary breasts.[6, 15, 24, 26, 30, 31, 44-46, 52, 54]

Aberrant breast tissue is dealt with by several authors. Roberts[42] discusses its recognition and treatment. Boas[5] describes the occurrence of accessory breasts in a study of 6,456 breast cases. Roux[43] reported the case of a lactating woman with bilateral axillary breasts in which milk exuded from a pore of the skin when pressure was made over the right mamma.

The pathology which may occur in aberrant breast tissue is dealt with by Uriburu and Marino,[53] who discuss dysplasia; Stringa[51] deals with tumours; Duany[16] gives an account of primary and secondary epithelial tumours of mammary structure in the axilla. Andersch[2] described cancerous degeneration in his studies on tumour formation in aberrant breast tissue. An additional case of carcinoma arising in presternal aberrant mammary tissue was reported by Maconi.[33]

The clinical material now reported consists of 11 cases, 9 of which presented as aberrant breast tissue and 2 as supernumerary breasts. All were axillary in location and all were females.

These 11 cases occurred in private practice in a series of 805 persons referred with breast conditions from 1934 to 1957. No operation was carried out for the accessory mammary tissue. The youngest patient was a girl of 12 just arrived at puberty. The oldest was a woman aged 47 who was menopausal. The average age was 33.3 years. Two patients were unmarried, 9 were parous women.

CLINICAL PICTURE

Supernumerary breasts may or may not function.

A 32-year-old woman in this series had a breast in each axilla, the size of a golf ball, complete with nipple and areola. They were painful when suckling and secreted milk.

A 12-year-old girl whose breasts were just developing had a small nipple below the left breast. Accessory mammary tissue was palpable beneath the nipple.

Aberrant Breast Tissue. Its varied situations have been described above. Such tissue may consist mainly of fat with a small amount of vestigial breast tissue hidden in it. Attention is usually drawn to such collections at puberty or when pregnancy or lactation occurs. These vestiges may or may not function. The oozing of milk from skin pores in some cases of aberrant axillary breast tissue has been reported. This is ascribed to the fact that the breast is developed from modified sebaceous glands, perhaps of apocrine type.

Choyce[8] quotes the statement of Champneys that during pregnancy the axillary sebaceous glands frequently enlarge and that occasionally true milk may be expressed from these hypertrophied glands through skin pores. So, too, Schreiner[48] speaks of the 'cutaneous gland of the axilla' which he described as another type of aberrant mammary tissue. He goes on to say that it is apparently a differentiation from the apocrine glands present in the axilla, which, at the onset of lactation, responds by enlargement and secretion, which continues for a week or so and then becomes quiescent.

These aberrant breasts feel like lipomas, especially in the axilla. Lipomas are extremely rare in this situation. de Cholnoky[13] stresses their rarity: in 100,000 admissions to the New York Post-Graduate Hospital no case of axillary lipoma was found.

On the balance of reasonable probabilities a fatty tumour in the armpit is therefore an accessory breast. Aberrant mammary tissue located in the axilla lies close to the skin. Glands are more deeply situated. Cystic degeneration, fibroadenomata and especially carcinoma may occur in aberrant breast tissue. The great malignancy of the latter has been alluded to above.

Many diagnostic difficulties may arise.

Of the 9 cases of aberrant axillary breast tissue here reported, 2 were unsuspected by patient or doctor. One was found on clinical examination in a lactating 33-year-old woman. It was the size of a walnut, tender, and distinct from the axillary tail, lying just under the anterior axillary fold.

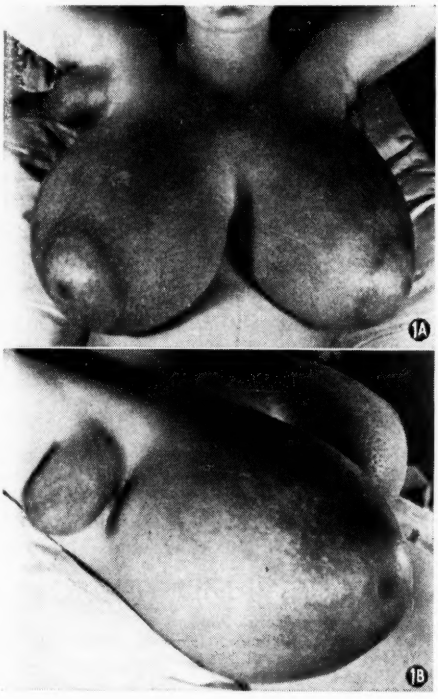
The second case was the 24-year-old wife of a doctor. She was followed up for 27 years. There was a mass 5 × 3 cm. in the left axilla. The breasts were the site of gross nodular fibroadenosis associated with considerable pain by which the aberrant mass was also affected. It enlarged during menstruation or lactation which the patient attributed to enlarged 'glands.' She was one of 3 cases in the series who felt so much discomfort in the affected axilla on carrying parcels that it was necessary to hold the arm(s) abducted.

Of the remaining 7 cases of aberrant axillary breast tissue 6 complained of pain there at the time of the monthly flow and were aware of a lump(s) which they had known of for periods varying from 5 days to many years. The masses in these 7 cases varied from case to case ranging from the size of a large walnut to that of an orange. To the latter group belonged a 47-year-old 4-para whose swellings were bilateral and symmetrical. An interesting case was that of a 33-year-old married woman with 3 children, the youngest aged 7 years. During each pregnancy a mass appeared in the right armpit, and became the size of a large orange during lactation. It was not painful, but awkward, as she had to keep the arm abducted. The mass disappeared when the children were weaned. There was no seepage from the skin and presumably there existed a communication with the duct system of the breast proper. When seen 7 years after the birth of the last child, no clinical evidence of accessory breast tissue was detectable in the axilla.

An unusual case is illustrated in Fig. 1. It concerns a married woman aged 28, referred to me in 1946 by Dr. Cort of Delareyville. She had a large mass of aberrant breast tissue in the right axilla and a smaller one in the left. She had a child aged 2 years.

After the baby was weaned her chest measurement across the bust was 32 inches. When I saw her the measurement was 51 inches. The aberrant breasts doubled in size

during her pregnancies and got smaller when lactating. When first seen she was 7 months' pregnant. She suffered from massive hypertrophy of both breasts which I removed without incident.



I have not seen the association of massive hypertrophy with the co-existence of bilateral aberrant axillary breast tissue mentioned in the literature.

TREATMENT

No treatment is necessary unless suspicion is aroused about the nature of the mass, in which case diagnosis must be made by biopsy. If carcinoma is detected, radical surgery is required.

In no case in this series was operation resorted to and in no case did the discomfort or cosmetic appearance induce the patient to request surgery.

CONCLUSIONS

Accessory breast tissue on or off the milk line which has a nipple with or without areola, i.e. supernumerary breast tissue, is common. It is not more liable to carcinoma than is the normally situated mamma. A large supernumerary breast in the labium or at the root of the thigh may require removal because of its nuisance value, otherwise surgery is rarely required unless a pathological process develops.

Aberrant breast tissue is the name given to accessory breast tissue which has neither nipple nor areola and which may be situated on or off the milk line. It is relatively uncommon and is a menace to the patient because it is prone to malignant degeneration. If this occurs the prognosis is grave despite radical surgery.

SUMMARY

1. The literature of congenital breast anomalies is reviewed.

2. These abnormalities are defined collectively as accessory breast tissue which consists of 2 groups:

(*a*) Supernumerary—common and not prone to cancer; and

(*b*) Aberrant—rare and liable to malignancy.

APPENDIX

Since going to press an additional case of aberrant breast tissue has been found in my records. It concerns a married woman of 23 years who was checked up yearly for 20 years. Both breasts were the site of diffuse nodular fibroadenosis for which no treatment was necessary. During the years of observation she had 3 children.

Three years ago a mass was found in the left axilla. Her breasts were small and this mass was well clear of the normal mamma. It was flat near the skin and left lipomatous and nodular. It was removed and reported on as accessory breast tissue containing 2 fibroadenomatous nodules.

The case is of interest in that, despite 3 pregnancies, accessory breast tissue was not detected until fibroadenosis occurred in the remnant; furthermore, it was the only case in the series which presented an indication for surgery.

REFERENCES

1. Ackerman, L. V. (1953): *Surgical Pathology.* London: Henry Kimpton, Ltd.
2. Andersch, H. (1958): Zbl. Chir., **83**, 1871.
3. Andreasen, A. T. (1948): Brit. J. Surg., **35**, 322.
4. Biancheri, T. (1932): Pathologica, **24**, 401.
5. Boas, H. (1955): Hautarzt. Zeitschr. für Dermatologie Venereologie und Verwante Gebiete, Berlin, Germany, **6**, 253.
6. Boas, H. (1956): Ugesk. Laeger., **118**, 518.

These 11 cases occurred in private practice in a series of 805 persons referred with breast conditions from 1934 to 1957. No operation was carried out for the accessory mammary tissue. The youngest patient was a girl of 12 just arrived at puberty. The oldest was a woman aged 47 who was menopausal. The average age was 33.3 years. Two patients were unmarried, 9 were parous women.

CLINICAL PICTURE

Supernumerary breasts may or may not function.

A 32-year-old woman in this series had a breast in each axilla, the size of a golf ball, complete with nipple and areola. They were painful when suckling and secreted milk.

A 12-year-old girl whose breasts were just developing had a small nipple below the left breast. Accessory mammary tissue was palpable beneath the nipple.

Aberrant Breast Tissue. Its varied situations have been described above. Such tissue may consist mainly of fat with a small amount of vestigial breast tissue hidden in it. Attention is usually drawn to such collections at puberty or when pregnancy or lactation occurs. These vestiges may or may not function. The oozing of milk from skin pores in some cases of aberrant axillary breast tissue has been reported. This is ascribed to the fact that the breast is developed from modified sebaceous glands, perhaps of apocrine type.

Choyce[8] quotes the statement of Champneys that during pregnancy the axillary sebaceous glands frequently enlarge and that occasionally true milk may be expressed from these hypertrophied glands through skin pores. So, too, Schreiner[18] speaks of the 'cutaneous gland of the axilla' which he described as another type of aberrant mammary tissue. He goes on to say that it is apparently a differentiation from the apocrine glands present in the axilla, which, at the onset of lactation, responds by enlargement and secretion, which continues for a week or so and then becomes quiescent.

These aberrant breasts feel like lipomas, especially in the axilla. Lipomas are extremely rare in this situation. de Cholnoky[13] stresses their rarity: in 100,000 admissions to the New York Post-Graduate Hospital no case of axillary lipoma was found.

On the balance of reasonable probabilities a fatty tumour in the armpit is therefore an accessory breast. Aberrant mammary tissue located in the axilla lies close to the skin. Glands are more deeply situated. Cystic degeneration, fibroadenomata and especially carcinoma may occur in aberrant breast tissue. The great malignancy of the latter has been alluded to above.

Many diagnostic difficulties may arise.

Of the 9 cases of aberrant axillary breast tissue here reported, 2 were unsuspected by patient or doctor. One was found on clinical examination in a lactating 33-year-old woman. It was the size of a walnut, tender, and distinct from the axillary tail, lying just under the anterior axillary fold.

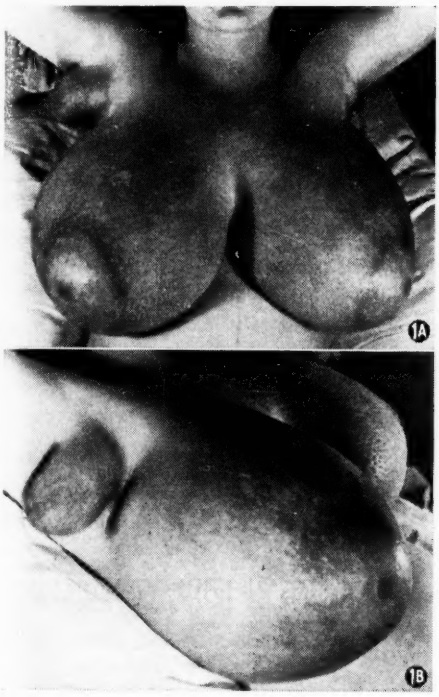
The second case was the 24-year-old wife of a doctor. She was followed up for 27 years. There was a mass 5 × 3 cm. in the left axilla. The breasts were the site of gross nodular fibroadenosis associated with considerable pain by which the aberrant mass was also affected. It enlarged during menstruation or lactation which the patient attributed to enlarged 'glands.' She was one of 3 cases in the series who felt so much discomfort in the affected axilla on carrying parcels that it was necessary to hold the arm(s) abducted.

Of the remaining 7 cases of aberrant axillary breast tissue 6 complained of pain there at the time of the monthly flow and were aware of a lump(s) which they had known of for periods varying from 5 days to many years. The masses in these 7 cases varied from case to case ranging from the size of a large walnut to that of an orange. To the latter group belonged a 47-year-old 4-para whose swellings were bilateral and symmetrical. An interesting case was that of a 33-year-old married woman with 3 children, the youngest aged 7 years. During each pregnancy a mass appeared in the right armpit, and became the size of a large orange during lactation. It was not painful, but awkward, as she had to keep the arm abducted. The mass disappeared when the children were weaned. There was no seepage from the skin and presumably there existed a communication with the duct system of the breast proper. When seen 7 years after the birth of the last child, no clinical evidence of accessory breast tissue was detectable in the axilla.

An unusual case is illustrated in Fig. 1. It concerns a married woman aged 28, referred to me in 1946 by Dr. Cort of Delareyville. She had a large mass of aberrant breast tissue in the right axilla and a smaller one in the left. She had a child aged 2 years.

After the baby was weaned her chest measurement across the bust was 32 inches. When I saw her the measurement was 51 inches. The aberrant breasts doubled in size

during her pregnancies and got smaller when lactating. When first seen she was 7 months' pregnant. She suffered from massive hypertrophy of both breasts which I removed without incident.



I have not seen the association of massive hypertrophy with the co-existence of bilateral aberrant axillary breast tissue mentioned in the literature.

TREATMENT

No treatment is necessary unless suspicion is aroused about the nature of the mass, in which case diagnosis must be made by biopsy. If carcinoma is detected, radical surgery is required.

In no case in this series was operation resorted to and in no case did the discomfort or cosmetic appearance induce the patient to request surgery.

CONCLUSIONS

Accessory breast tissue on or off the milk line which has a nipple with or without areola, i.e. supernumerary breast tissue, is common. It is not more liable to carcinoma than is the normally situated mamma. A large supernumerary breast in the labium or at the root of the thigh may require removal because of its nuisance value, otherwise surgery is rarely required unless a pathological process develops.

Aberrant breast tissue is the name given to accessory breast tissue which has neither nipple nor areola and which may be situated on or off the milk line. It is relatively uncommon and is a menace to the patient because it is prone to malignant degeneration. If this occurs the prognosis is grave despite radical surgery.

SUMMARY

1. The literature of congenital breast anomalies is reviewed.

2. These abnormalities are defined collectively as accessory breast tissue which consists of 2 groups:

(*a*) Supernumerary—common and not prone to cancer; and

(*b*) Aberrant—rare and liable to malignancy.

APPENDIX

Since going to press an additional case of aberrant breast tissue has been found in my records. It concerns a married woman of 23 years who was checked up yearly for 20 years. Both breasts were the site of diffuse nodular fibroadenosis for which no treatment was necessary. During the years of observation she had 3 children.

Three years ago a mass was found in the left axilla. Her breasts were small and this mass was well clear of the normal mamma. It was flat near the skin and left lipomatous and nodular. It was removed and reported on as accessory breast tissue containing 2 fibroadenomatous nodules.

The case is of interest in that, despite 3 pregnancies, accessory breast tissue was not detected until fibroadenosis occurred in the remnant; furthermore, it was the only case in the series which presented an indication for surgery.

REFERENCES

1. Ackerman, L. V. (1953): *Surgical Pathology.* London: Henry Kimpton, Ltd.
2. Andersch, H. (1958): Zbl. Chir., **83**, 1871.
3. Andreasen, A. T. (1948): Brit. J. Surg., **35**, 322.
4. Biancheri, T. (1932): Pathologica, **24**, 401.
5. Boas, H. (1955): Hautarzt. Zeitschr. für Dermatologie Venereologie und Verwante Gebiete, Berlin, Germany, **6**, 253.
6. Boas, H. (1956): Ugesk. Laeger., **118**, 518.

7. Bresslau, E. (1920): *The Mammary Apparatus of the Mammalia.* London: Methuen and Co.
8. Choyce, C. C. (1932): *A System of Surgery,* 3rd ed., Vol. 1, p. 954. London: Cassell and Co., Ltd.
9. Christopher, F. (1936): *A Textbook of Surgery,* p. 926. Philadelphia and London: W. B. Saunders Co.
10. Cole, W. H. (1948): In *Practice of Surgery,* Dean Lewis. *Breast Section,* Vol. 5, Chapter 6, p. 7. Hagerstown, Md.: Prior Co., Inc.
11. Deaver, J. B. and McFarland, J. (1917): *The Breast.* Philadelphia: P. Blakiston, Son and Co.
12. de Cholnoky, T. (1939): Arch. Surg., **39,** 926.
13. de Cholnoky, T. (1951): New York State J. Med., **51,** 2245.
14. Della Romana de Compte, L. and Radice, J. C. (1957): Obstet. Ginecol. Latino-Americanos, **15,** 203.
15. Dexter, M. W. and H. L. T. (1955): South Med. J., **48,** 1187.
16. Duany, N. Puente (1955): Arch. Cubanos Cancerol., **14,** 143.
17. Dubrausky, V. (1960): Zbl. Gynaek., **82,** 558.
18. Erdman, J. F. (1934): Med. Rec., **140,** 583.
19. Falk, V. S. (1950): Minerva Med. J., **49,** 1007.
20. Fenech, H. B. (1949): Harper Hosp. Bull., **7,** 268.
21. Francheschini, P. (1947): Sez. Prat., **54,** 380.
22. Geschickter, C. F. (1943): *Diseases of the Breast.* Philadelphia and London: J. B. Lippincott Co.
23. Giacobbe, C. (1930): Minerva Med. J., **2,** 834.
24. Giocoli, Nacci (1955): Gior. Ital. Chir., **11,** 130.
25. Gomes, F. P. (1958): Med. Contemp., **76,** 409.
26. Grcic, R. Z. J. (1959): Srpski Arhiv. za Celokupno Lekarstvo, **87,** 953.
27. Guest, E. (1923): *Correspondence,* Brit. Med. J., **2,** 85.
28. Haagensen, C. D. (1956): *Diseases of the Breast.* Philadelphia and London: W. B. Saunders Co.
29. Hendrix, R. C. and Behrman, S. J. (1956): Obstet. and Gynecol., **8,** 238.
30. Kowalski, M. S. (1958): Pathologia Polska, **9,** 73.
31. Leckie, F. H. (1951): Brit. Med. J., **1,** 1060.
32. Looney, C. M., Reichman, S. C. and Noel, O. F. (1959): Amer. Surgeon, **10,** 219.
33. Maconi, G. (1954): Riforma Med., **68,** 589.
34. Masor, N. (1952): New York State Med. J., **52,** 1674.
35. Massabuau, G. A. and Gilbert, H. (1933): Bull. Soc. d'Obst. et de Gynéc., **22,** 765.
36. Matti, H. (1936): Schweiz. Med. Wchnschr., **17,** 1152.
37. Noronha, A. J. (1936): Brit. J. Surg., **24,** 143.
38. Purves, R. and Hadley, J. A. (1927): Brit. J. Surg., **15,** 279.
39. Ravdin, J. S. (1934): Surg. Clin. N. Amer., **14,** 139.
40. Raven, R. W. (1960): *Cancer.* London: Butterworth and Co., Ltd.
41. Razemon, P. and Bizard, G. (1929): Rev. de Chir., **67,** 226.
42. Roberts, J. M. (1952): West J. Surg., **60,** 175.
43. Roux, J. P. (1955): Brit. Med. J., No. 4904, p. 28.
44. Ruggiero, J. R. (1957): Dia Med. B. Air., **29,** 800.
45. Sabbatini, C. (1952): Riforma Med., **66,** 945.
46. Salvetti, M. E. and Scavo, R. (1955): Chir., Ital., **8,** 3.
47. Schmidt, T. and Tannwald, W. J. (1927): Zentralb. f. Gynak., **51,** 917.
48. Schreiner, B. F. (1944): In *Christopher's Textbook of Surgery,* 3rd ed., p. 890. Philadelphia and London: W. B. Saunders Co.
49. Siegler, A. L. and Gordon, R. (1951): Amer. J. Obstet. Gynecol., **62,** 1367.
50. Slepykh, A. S. (1959): Akusherstvo I Ginekologiia, **35,** 51.
51. Stringa, U. (1951): Minerva Chir., **6,** 349.
52. Uriburu, J. V., Jr. and Yoel, J. (1950): Prensa Med. Argent., **36,** 2398.
53. Uriburu, J. V. and Marino, E. (1952): Prensa Med. Argent., **39,** 2961.
54. Winkler, E. (1958): Bruns Beiträge zur Klinischer Chirurgie, **197,** 24.

'SHOCK,' HYPOTENSIVE DRUGS AND 'HYPOTENSION ANAESTHESIA'

H. H. SAMSON, *M.B.E.,* M.R.C.S., L.R.C.P.
Johannesburg

In clinical medicine one must guard against the fallacy of arguing *post hoc, ergo propter hoc.* Unfortunately, the assumptions, postulations and deductions on which such hypotheses are based, though pseudo-scientific, are often plausible and fall on many sympathetic ears.

The validity of a treatment, or the effects of a drug, can sometimes be verified at autopsy; but this is hardly possible with an unfavourable outcome to a clinical condition such as 'shock' and 'anaesthetic coma' (general anaesthesia).

The apparent mystery which enshrouds these highly controversial subjects, is due to the mystery surrounding the associated deaths. Fatalities result from either hypoxia and hypercapnia, or anoxia. As yet, there are no conclusive post-mortem findings by which death resulting from such grave physiological changes can be substantiated.[1]

While other members of the profession can learn from the dead, clinicians (and these include anaesthetists) can only become proficient

by making observations on the living. One cannot, therefore, accept readily what must be regarded as an entirely new concept of the physiology of the circulation of the blood, and what naturally must follow—an entirely new approach to clinical medicine.

It is now common knowledge that there are many anaesthetists and other clinicians all over the world, who believe that 'shock' can not only be prevented, but may successfully be treated with hypotensive drugs. Patients anaesthetized with 'hypotension anaesthesia' are better 'protected'[2, 3] against 'shock' than if anaesthetized by conventional methods; that what is considered to be safe, is ordinary anaesthesia supplemented with hypotensive drugs, epidural and spinal anaesthetics, and narcosis effected with that potent drug, halothane (a useful anaesthetic agent if not 'pushed'). It is also postulated that since vasoconstriction is the cause of all evil, it should be prevented, especially when 'shock' tends to become irreversible, and treated with hypotensive drugs.[2, 3]

There are also some who feel that it is now necessary to challenge the validity of the all-important physiological entity, viz. the critical systolic blood pressure level.[3] Apparently this has arisen from observations which were made during 'hypotension anaesthesia' where no untoward side effects were demonstrated. (In many instances, the blood pressures were unrecordable by either palpation or auscultation).[3] The coma was maintained by ether in the presence of approximately 100% oxygen, and all the patients were allowed to breathe unaided.[3]

Noradrenaline types of vaso-pressor drugs are strictly forbidden. It is assumed that since 'shock' is a state of vasoconstriction, the heart would be subjected to unnecessary strain. It is also assumed that everything is activated for the benefit of the heart, which is the vital factor in the maintenance of life.[3]

It is suggested that it would be a good thing to treat coronary thrombosis associated with 'shock' with hypotensive drugs.[3]

In this enlightened era of medicine, it is incredible that there are doctors who have acquired such an approach to clinical medicine —this naturally includes the giving of an anaesthetic. It is felt that their 'highly satisfied' attitude with the outcome to their patients, who were subjected to anaesthesia and 'shock,' can only be ascribed to the fact that 'words do not mean what you think they mean. Sincerity is relative and self deception prevalent.'

Unless an immediate halt is called to this new conception of medical principles, not only is general anaesthesia liable to fall into disrepute as a science, but also medicine as a whole. Furthermore, it would appear opportune to emphasize that a subject is not dead merely with cessation of the beating of the heart. Death only occurs when the brain has been deprived of oxygen for 4-5 minutes, and everything must be done to supply the brain with the oxygen which it needs—if by chance the heart happens to stop.

Many will agree that the term 'shock' is a centuries-old euphemism for *the clinical state of hypoventilation associated with acute circulatory failure,* a state which occurs when the normal physiological factors which are responsible for maintaining an adequate circulation of the blood become deranged.

These factors are the two axiomatic physiological entities—the *tidal volume* and the *critical systolic blood pressure level.* They are responsible for providing an adequate gaseous exchange between the blood and the alveolar air. *They are as inseparable from each other as the respiratory system is from the cardiovascular system.* When these all-important entities are interfered with and depressed, the vital nuclei in the hypothalmus and the vital centres in the medulla, such as the vasomotor centres and the respiratory centre, are deprived of oxygen and may also be subjected to the poisonous effects of CO_2. They are then unable to function effectively, i.e. to provide an adequate ventilation and the adequate blood pressure essential for an adequate circulation. If such a state is not corrected, it inevitably leads to hypoxaemia and, as mentioned, carbon dioxide intoxication (hypercapnia).

The subject, who may have previously been in a physiological state, can now suddenly be transferred into an unphysiological and potentially dangerous clinical state known as hypoxia and hypercapnia, in other words—*'shock.'*

This clinical state is likely to occur no matter what caused the tampering with the physiological entities. Thus while the *propter hoc* cause of 'shock' may vary, the *ad hoc* cause is always the same.

A subject in 'shock' can die from severe hypoxia before CO_2 has a chance to take effect. He may recover spontaneously, and unaided, if the condition is not severe; or he may be subjected to a long period during which the functions of brain and other important organs nearly as vital as the brain, are slowly being destroyed by lack of oxygen and the poisonous effects of CO_2. When the brain has been so damaged, in spite of all known treatments, the circulation of the blood is not able to be maintained adequately, the condition

can then be said to have become incurable or *irreversible.* If recovery has been made possible before irreversibility, and the parenchymatous organs have not been seriously affected, the subject may regain his former healthy state; but if the vital organs have lost their normal function, as a result of hypoxaemia and hypercapnia, the subject may linger on for days and even weeks finally to succumb to the grave effects of acidaemia. This is the inevitable outcome when the acid-base mechanism is unable to withstand the serious assaults which are made on the biochemistry of the blood by serious physiological changes.

Anaesthetists, in general, are fully aware that what has been written here may well occur in general anaesthesia, or if epidural and spinal anaesthetics are used to abolish pain. It is known, only too well, that although an anaesthetic mixture can be richly augmented with oxygen, 'shock' (known as post-operative collapse) can easily become manifest; that while the nature of the operation (blood loss, pain, etc.) and even the surgeon may have to be indicted, 'shock,' or collapse, associated with surgery, is more often than not due to 'anaesthetic collapse.'[4] The finger therefore has to be pointed to the *Man Behind The Bag.*[5]

Frequently, the ventilation of the patient is inadvertently neglected. If the breathing does require assistance or control, insufficient precautions are taken to ensure adequate CO_2 elimination. The latter is often caused by incorrect pumping of the re-breathing bag. Inadequate abolition of painful stimuli is another cause of post-operative 'shock.'

Most anaesthetists are also aware that the tidal volume and the critical systolic blood pressure of a subject under basal conditions, in the prone position, are about 350-500 c.c. and 60-70 mm. of Hg respectively; but they are liable to greater variations in pathological conditions, e.g. in severe hypertension, the critical systolic blood pressure level may be as high for that individual as the normal systolic pressure of an healthy subject, while the critical level of a tidal volume for an acclimatized emphysematous patient may be actually lower than the lower level of a tidal volume in a child. Therefore, when dealing with unhealthy patients, anaesthetists are often obliged to break certain medical principles. But generally every precaution is taken to keep the anaesthetized patient *as near to his physiological state as possible.* This is done not only to avoid 'shock,' but to avoid a far more serious complication, viz. the clinical state of anoxia (cardiac arrest) known as syncope (the Greek for cutting off), but only as a fainting attack to many. There is an even graver condition which all practitioners try to prevent—the terrible state of a human vegetable.

It must now be evident that when the term 'shock' is used in medical circles, it can only infer the clinical state of hypoventilation. Furthermore, this is the only basis in which 'shock' can be considered. As mentioned before, the mechanism which controls the normal circulation becomes less effective as a result of inefficient vasomotor centres and a depressed respiratory centre. Thus the blood vessels lose their normal state of slight tension (this tone is axiomatic because the vasopressor effect of the vasomotor system predominates slightly over the vasodilator). Besides this loss of tone in the blood vessels, the important splanchnic nerves are directly affected. As a result, the splanchnic area becomes a huge reservoir for stagnant blood. To a lesser extent, it is assisted by the arterioles in the muscles, and the brain, and the arteries and veins, having lost their tone, are now widely dilated.

A 'shocked' subject lies quietly in a subdued state. Even the untrained eye of a casual observer can correctly judge how little extra would be necessary to cause death. The severity of such a state depends on the factors which usually apply to clinical conditions in general: acuteness of onset; severity of depression of the brain, duration, and the all-important compensatory mechanism peculiar to different persons.

In a moderate case of 'shock,' however, the reflexes and all the ordinary functions are in a subnormal state. The subject may be unconscious, but if he talks, he is incoherent. He is no longer garrulous, anxious, restless or sweating, symptoms which may have occurred if the higher centres were subjected to hypoxaemia and stimulated, or a full bounding pulse due to CO_2 intoxication. The subject does not now complain of pain if there is pain. The skin is pale and will bleed if damaged (*cf.* syncope, where the skin is still warm and pallid). The veins, which are readily seen in syncope (there has been insufficient time for the splanchnic area to take up much blood), cannot be easily found in 'shock' because of their collapsed state. The pulse is rapid and thready. The pupils may be of any size, varying with the light. They are, however, usually smaller than if the subject has not been in 'shock.' The pupils can become very dilated in sudden and severe 'shock,' but they are never as dilated as in cardiac arrest, and the reaction to light is sluggish.

In cardiac arrest, the pupils *suddenly* become completely dilated. This dilation is symptomatic of acute oxygen want and of massive production of noradrenaline as well. Inciden-

tally this noradrenaline discharge is probably the cause of the sudden vasoconstriction of the arterioles in the skin, giving the skin a characteristic pallor. Since both these centres are rendered powerless by the acute anoxaemia, the pupils gradually become smaller until death, when they are neither dilated nor constricted—the light reflex is no longer a variable.

In syncope, the vasoconstriction of the arterioles in the skin is relieved. The anoxaemic blood from the general circulation now enters the capillaries and discolours the skin, giving it the characteristic blotchy appearance. However, in 'shock,' vasoconstriction of the skin is a terminal sign, and the skin first becomes cyanosed.

When treating 'shock' it must be remembered that since the *ad hoc* cause is the breaking down of the physiological entities, these must be restored to normal forthwith. This consists mainly of providing the subject with an *adequate ventilation* and an *adequate blood pressure.*

It may be necessary to assist the breathing manually with a re-breathing bag in the presence of a high oxygen flow, with or without a carbon dioxide absorber. A tracheotomy[6] may be necessary, or even the services of an iron 'lung.'

In as much as the *ad hoc* cause needs urgent attention, so must it be remembered that there may be many grave *propter hoc* factors which require equal and as urgent consideration. Antidotes, such as anti-snakebite serum, insulin, sugar, etc. may have to be given (and repeated many times) to combat the grave and sometimes fatal effects of the poisons on the blood chemistry. They may also have to be administered to prevent further poisoning of the blood and tissues. In a severe CO_2 poisoning, the entire blood volume may have to be removed and replaced with *fully oxygenated blood.* Where blood loss is the cause of 'shock,' it should be restored quickly; but if blood is not available, the depleted circulation should be made good with plasma-like fluids in order that the heart can operate more effectively as a pump. Intractable pain (a common and potent instigator of 'shock') should be relieved as soon as possible. If morphine cannot control pain in ordinary doses, it may be necessary to accomplish this with a general anaesthetic. This should be done in severe traumatic 'shock,' especially when there has been loss of blood as well. Such a subject, if not treated immediately because of a dangerously low blood pressure, may never reach the operating theatre. The question whether he has had food is of no importance. In severe shock there is often reversed peristalsis of the bowels due to the vasomotor upset. As a result, vomiting can occur as long as there is *any* food left in the intestines. Very often even liquid faeces are regurgitated. Such a case needs the services of a skilful anaesthetist who can pass an endotracheal tube quickly and pack off the opening of the larynx. But it is a *sine qua non* that the ventilatory requirements of such a patent must be maintained from then onwards until the effects of the narcotic have worn off, the pain relieved, and the normal complement of the circulation restored, i.e. until the clinical state of hypoventilation has been overcome, if possible.

In order to restore a reasonable blood pressure, the administration of a vasoconstrictor drug of the *noradrenaline* type may be necessary. It is essential to use this type of vasopressor drug which is capable of constricting *all* arterioles. This cannot be effected by adrenaline, which has no pressor effect on the arterioles in the muscles and the brain. It is therefore absolutely of no use in severe 'shock' where the stagnation of the blood throughout the body, including the brain, is the predominant feature.

It is incorrect to suggest that the 'straining' of the heart in 'shock' is caused by vasoconstriction. In the first place, there is no vasoconstriction and, secondly, this mighty organ can well look after its own welfare—it is known to be able to withstand approximately 9 times the ordinary strain to which it is accustomed, and probably a little less as a result of myocardial infarction. It should be realized that pain is often the most important cause of 'shock' in coronary thrombosis. If the condition, however, is 'silent' and severe, all that is required of the vasopressor drug is to restore a little head of pressure so as to shift the sluggish circulation. In any case, the patient is probably lying prone, or in the Trendelenburg position. In view of the suggested treatment of hypotensive drugs for 'shock of all kinds,' it would be interesting to learn how a victim of a bite from a snake such as a black mamba would fare with this type of therapy. It is known that this venom has a similar predilection for the sympathetic nervous system as have the hypotensive drugs; that the blood pressure will drop as rapidly when the poison enters the general circulation—perhaps even a little faster than with a hypotensive drug; but that it is virtually impossible to remove the poison from the nervous tissues. Added to this hazard, there is another, even if not so great, viz. the curarizing effect of the venom on

muscles. Indeed, there are probably few graver forms of 'shock' than this. May it be suggested that wherever black mamba snakes choose to strike, intended victims ask for Levophed instead of Arfonad to augment their anti-snake bite outfits.

One would also like to learn what doses of hypotensive drugs are suggested and what new low level of the blood pressure is required to alleviate 'strain' in other shocking conditions such as hypo- or hyperglycaemic coma, or 'shock' due to altitude sickness; and, what is the main concern of those who administer anaesthetics, post-anaesthetic collapse and blood loss.

In spite of all this, the medical profession is led to believe that the blood pressure can be dropped with impunity to unrecordable levels, and that no morbidity of any description will be demonstrated. Many will agree, and especially anaesthetists, that if this is the case, even for the removal of a scar, then it is possible that pigs could fly—without wings! If a subject, no matter how fit, is subjected to this type of anaesthesia, for say one hour, and does not spend at least another hour in the recovery room (one usually recovers in the ward from a surgical assault) one would also be more than surprised.

There will be many who might find it extremely difficult to accept that patients treated with hypotensive drugs for 'shock' must owe their recovery to these drugs. It will be felt that those who had recovered, did so in spite of this new form of treatment.

Arfonad, however, has its uses outside general anaesthesia. It can be put to great advantage in severe asthma. When the blood pressure has risen alarmingly, it may still be necessary to use a more potent drug than adrenaline by which to relieve intractable bronchiolar spasms. This naturally, is not without danger. To combat a possible cerebral or myocardial accident (the heart may be diseased), the judicious use of Arfonad is recommended.

To administer hypotensive drugs and/or to deprive a patient in 'shock' of noradrenaline must be regarded as rank malpractice.

Many will agree that, however skilful and alert an anaesthetist is, it is axiomatic to squeeze the re-breathing bag, even periodically and especially when the blood pressure is subnormal or below critical levels; otherwise the anaesthetist may as well be with the patient's relatives hoping that all's well in the operating theatre.

If there are any more who still believe that pigs can fly, it is suggested that another type of axiom be tampered with, viz. 'Do unto others as you would have them do unto you.' But this is not a safe 'idea.' A general anaesthetic can be supersaturated with oxygen to the extent of nearly 100%. In the ambient atmosphere, however, there is never more than 20.83%.

Those who are obliged to administer 'hypotensive anaesthesia' for surgeons who feel that a 'bloodless field' is essential for certain types of operations, are no doubt rendering a service. Nevertheless, it must be emphasized that in order for the patient not to be unduly subjected to the hazards of this potentially dangerous anaesthesia, it will be necessary to pay just a little more attention to the ventilatory and blood pressure requirements than is customary with conventional methods.

Finally, let it be stated that the whole field of scientific endeavour and the over-all safety to human beings has been immeasurably enhanced by the courage of those investigators who elect to make observations on human beings. This has, however, not resulted from courage alone, but because those observers take every precaution to practise their medicine and conduct their anaesthetics to the best of their ability on sound medical and ethical lines. The subject should not be harmed or poisoned in determining the effects of a drug or of a technique. Once the drug or the technique becomes uncontrollable, i.e. beyond that required to produce the desired effects, and the physiology is neglected, the observations made do not reflect the true effects of the drug or the technique *per se*. Instead, they reflect the unwitting poisoning of the subject (although he may be a volunteer or an unfortunate animal), and serve only to provide a mass of wierd toxicological phenomena which should be unacceptable in any branch of medicine. Such pseudo-scientific research is done by those apparently untrained in the intricacies of physiological experimentation—the 'anaestheto-physiologist' is often guilty of this and is responsible for much of the unnecessary mystery which enshrouds clinical medicine, viz. the 'may-be's' and the 'might-be's' which abound in 'shock' and general anaesthesia.

REFERENCES

1. Gordon, I. (1944): *A Classification of Deaths of Medico-Legal Importance*, Brit. Med. J., **2**, 337.
Shapiro, H. A. (1953): Editorial: *Is Asphyxia a Pathological Entity Recognizable Post-Mortem?* J. Forensic Med., **1**, 65.
2. Johnstone, M. (1961): Med. Proc., **7**, 228.
3. Bentel, H. (1960): Med. Proc., **6**, 333.
4. Samson, H. H. (1956): S. Afr. Med. J., **30**, 470.
5. Rumble, Jr., Lester (1956): South Med. J., April.
6. Elliott, G. A. *et al.* (1960): Med. Proc., **6**, 402.

MEGALOBLASTIC ANAEMIA DUE TO VEGANISM

H. C. SEFTEL, B.Sc., M.B., B.Ch., DIP. MED. (RAND)
Department of Medicine, Baragwanath Hospital, Johannesburg

A vegan, i.e. a strict vegetarian, restricts his diet to products of the plant kindom. He eats neither animal flesh nor any form of animal product such as milk, butter or eggs. He therefore, differs from the ordinary vegetarian whose diet does include dairy products. Since the plant foods normally consumed by man contain negligible amounts of vitamin B_{12},[1] vegans constitute an ideal experimental human group for studying the effects of dietary deficiency of vitamin B_{12}. In Great Britain some hundreds of ordinary vegetarians took to veganism after 1945 and have been well studied by Wokes *et al.*, whose findings are of considerable interest.[1, 2] About half the British vegans escaped serious illness, even after many years of veganism, and in some of these normal vitamin B_{12} levels suggested a normal vitamin B_{12} status. It was presumed that such persons obtained sufficient quantities of vitamin B_{12} from synthesis by bacteria inhabiting the intestinal tract or contaminating the ingested vegetable foods. In the other half, who developed signs of vitamin B_{12} deficiency, it might have been expected that these would follow the pattern found in the classical vitamin B_{12} deficiency disease, pernicious anaemia, the principal features of which are megaloblastic anaemia and neurological changes such as subacute combined degeneration of the cord. In actual fact the vegans rarely developed megaloblastic anaemia and even macrocytosis of pronounced degree was uncommon. Neurological changes, on the other hand, were marked and in 2 vegans death was attributed to these changes.

In this paper the case is recorded of an African vegan who, in contrast with the findings in British vegans, presented with megaloblastic anaemia without any evidence of neurological involvement.

CASE REPORT

D. D., an African male aged 50, was admitted to Baragwanath Hospital on 11 January 1960 complaining of breathlessness and swelling of the feet of 3 weeks' duration. Apart from an appendicectomy in 1956 the past history was negative.

He gave the following interesting personal history. Until 1949 his diet was unrestricted. At that time he began to study the Bible and in accordance with the commandment, *Thou shalt not kill*, decided that henceforth he would eat neither meat nor any other form of animal product such as milk, butter or eggs.

A typical day's diet consisted of the following:

Breakfast: Maize meal porridge, bread or scones with margarine, and tea without milk.

Lunch: Bread and tomato sandwiches, black tea, some form of fruit—usually an orange or an apple.

Supper: Maize meal porridge, vegetables, bread and black tea.

He ate a variety of vegetables, the commonest being potatoes, tomatoes and cabbage. He would eat these 3 vegetables about 4-5 times a week. Other vegetables such as pumpkin, carrots, peas, beans, spinach and onions were eaten about once weekly. All the vegetables except tomatoes were cooked before eating. The bread he ate was baked for him by his wife, since he believed that the commercial bakers added eggs or milk to their product.

The patient was by occupation a furniture polisher of 20 years' standing, earned R40 a month and had a wife and 6 children.

He was intelligent and co-operative. His state of nutrition was normal and the blood pressure was 130/70 mm. Hg. The mucous membranes were decidedly pale and there was moderate oedema of both ankles. The spleen was 2 fingers enlarged, and in the right iliac fossa an old appendicectomy scar was present. The central nervous and other systems were normal.

LABORATORY INVESTIGATIONS

Urinalysis was normal.

The haemoglobin level was 9.3 g. %, the haematocrit 26% and the mean corpuscular haemoglobin concentration 36%. The red cells showed marked anisocytosis and some poikilocytosis and schistocytosis. The reticulocyte count was 2%. Platelets were numerous. The white cell count was 4,800 per c.mm. (Neutrophils, 54%; lmyphocytes, 43%; monocytes, 3%). Examination of the bone marrow showed well-marked megaloblastic changes.

The serum vitamin B_{12} level (microbiological assay using *Lactobacillus leichmanii*) was 40 $\mu\mu$g. per ml. (normal range 140 to 900 $\mu\mu$g. per ml).

The augmented histamine test revealed the presence of free hydrochloric acid.

A barium meal and follow through revealed no abnormality of the gastro-intestinal tract.

The serum bilirubin was 1.3 mg. per 100 ml. and the blood urea 15 mg. per 100 ml. The Schumm's and Coomb's tests were negative.

An X-ray of the chest and the ECG were normal.

COURSE

In the ward the patient continued to adhere to what he thought was a strict vegetarian diet. However, unbeknown to him, a number of foods which he regarded as acceptable, such as cereal porridges, did in fact contain animal protein in the form of milk, cream, butter or eggs. This was not deliberate on our part but simply arose from the hospital custom of enriching certain plant foods by the addition of dairy products. Where, as in the case of cereal porridges, the addition was small in amount and intimately mixed, it escaped detection by the patient.

On 2 February, 3 weeks after admission, a repeat blood count revealed a reticulocytosis of 16% and a haemoglobin level of 9.6 g. %.

On 8 February the reticulocyte count was 4% and the haemoglobin 10.2 g. %.

A week later the haemoglobin had risen to 11.3 g. %. The anaemia was clearly improving and the improvement could only be attributed to the diet, since all haematinic therapy had been withheld.

In addition his oedema and dyspnoea had disappeared and he was feeling well.

On 28 February the patient was discharged on an oral dose of 10 μg. of vitamin B_{12} daily.

On 11 March 1960, when seen as an out-patient, his haemoglobin level was 15.8 g. % and the spleen was no longer palpable.

In June and December 1960 his haemoglobin levels were 15.9 g. % and 16.1 g. % respectively.

DISCUSSION

The features of this case leave no doubt that the megaloblastic anaemia was due to vitamin B_{12} deficiency resulting from an 11-year period of veganism. Two points require explanation.

i. There is the patient's haematological improvement in response to a hospital diet which could have contained only very small quantities of vitamin B_{12}. In a normally balanced diet the vitamin B_{12} content varies from 16.0 to 31.0 μg. per day, the major part of this being provided by meat of various types.[3] Dairy products contain relatively small amounts[4] (e.g. milk contains 0.4 μg. per litre) and it was these products taken in small quantities mixed with foods of plant origin, which constituted the main source of vitamin B_{12} in the patient's hospital diet. However, careful observations by Darby *et al.*[3] in vitamin B_{12}-deficient subjects with pernicious anaemia, have demonstrated that as little as 0.5 μg. of vitamin B_{12} daily is sufficient for both the induction and maintenance of a complete haematological remission. Darby *et al.* administered this minute dosage parenterally, but in subjects with normal gastric intrinsic factor like our African vegan, vitamin B_{12} is effective orally, and the amount needed to ensure the absorption of 0.5 μg. may be as little as 0.6 μg.[5]

We consider it reasonable to suggest that vitamin B_{12} in quantities of this order was, in fact, provided by the dairy products in our patient's hospital diet. It is also possible that the ingestion of the dairy products produced a change in the bacterial flora of the bowel which resulted in an increased intestinal synthesis and absorption of vitamin B_{12}.[1]

ii. The patient's presentation with megaloblastic anaemia without neurological involvement requires explanation. This is the exact reverse of the situation in British vegans, in whom anaemia is rare and the symptomatology is largely referable to the nervous system. These contrasting findings may possibly be related to differences in folic acid intake between our patient and the British vegans.

Folic acid is intimately linked with vitamin B_{12} in the maintenance of normal haemopoiesis and, particularly relevant here, is the evidence that in certain circumstances the relationship between the 2 substances may be reciprocal, the administration of large doses of the one tending to mobilize and deplete the body stores of the other.[6-8] Thus, to take the best documented example of this relationship, when large doses of folic acid are administered to cases of vitamin B_{12} deficiency with pernicious anaemia, it is found that haematological remission may ensue but that neurological manifestations such as subacute combined degeneration of the cord may be precipitated or exacerbated. These findings cannot be explained as an effect of big doses of folic acid directly on the haemopoietic and nervous system. The most reasonable hypothesis[8] is that the effects are mediated indirectly, the large doses of folic acid stimulating the mobilization to the bone marrow of what little vitamin B_{12} the pernicious anaemia patient still possesses. This brings about normal haemopoiesis but deprives the nervous system of vitamin B_{12}, thus precipitating or aggravating neurological changes.

The rarity of megaloblastic anaemia in British vegans may be due to the fact that folic acid is present in excess in their diet, which contains large amounts of fresh, green, leafy vegetables. This excess of folic acid would ensure that enough of the little vitamin

B_{12} which the vegans obtained from bacterial synthesis, would be directed towards maintaining normal haemopoiesis. If there were sufficient vitamin B_{12} left over when the demands of the bone marrow had been met, the functional integrity of the nervous system would be maintained, as is the case of about half of British vegans. Otherwise, as occurs in the other half, neurological symptoms develop.

The unusual presentation of our Bantu vegan with megaloblastic anaemia in the absence of neurological manifestations may, we suggest, be related to the possibility that the folic acid content of his diet was not excessive and may in fact have been low. His economic status was such that he was unable to purchase the large quantities of fresh, green, leafy vegetables commonly consumed by British vegans. The plant foods in his diet consisted largely of potatoes, tomatoes, cabbage, maize, oranges and apples, all of which have a relatively low folic acid content.[9, 10] Furthermore all the vegetables, apart from tomatoes, were eaten cooked and some of the folic acid may have been destroyed by heating.[11] In the presence of a low or normal folic acid intake, the stimulus to mobilization of his meagre stores of vitamin B_{12} to the bone marrow may have been lacking. This resulted in the development of megaloblastic anaemia but ensured that enough of the small amounts of vitamin B_{12} obtained from bacterial synthesis, was available for the maintenance of normal neural function.

Although exceptional, our African vegan is not unique. At least 2 other cases of megaloblastic anaemia due to dietary deficiency of vitamin B_{12} have been reported,[12, 13] and it is interesting that in one of these[12] the intake of folic acid was almost certainly not excessive. This was an old age pensioner who lived on a diet of bread, margarine, tea and potatoes. The other case[13] was a chronic, severe, paranoid schizophrenic who had been unemployed for 25 years before presenting with megaloblastic anaemia. Unfortunately, the dietary history as given in the case report, although vegan in type, is not sufficiently detailed to permit a reliable estimate of the folic acid intake.

SUMMARY

The case is described of an African male who developed megaloblastic anaemia due to vitamin B_{12} deficiency resulting from an 11-year period of veganism (strict vegetarianism).

Neurological manifestations were absent.

This picture is contrasted with that of British vegans in whom megaloblastic anaemia is rare and neurological signs common.

A possible explanation for this difference is suggested.

I wish to thank Dr. I. Frack, Superintendent of Baragwanath Hospital for permission to submit this case report for publication, and Dr. J. Metz, of the South African Institute for Medical Research, for the vitamin B_{12} assay.

REFERENCES

1. Wokes, F. (1956): Proc. Nutr. Soc., **15**, 134.
2. Wokes, F., Badenoch, J. and Sinclair, H. M. (1955): Amer. J. Clin. Nutr., **3**, 375.
3. Darby, W. J., Bridgforth, E. B., Le Brocquy, J., Clark, S. L., De Oliveira, J. D., Kevany, J., McGranity, W. J. and Perez, C. (1958): Amer. J. Med., **25**, 726.
4. Goodman, L. S. and Gilman, A. (1955): *The Pharmacological Basis of Therapeutics*, 2nd ed. New York: The Macmillan Company.
5. Mollin, D. L. (1959): Brit. Med. Bull., **15**, 8.
6. Goldsmith, G. A. (1958): Amer. J. Med., **25**, 680.
7. Narayanan, M. S., Shenoy, K. G. and Ramasarma, G. B. (1957): Indian J. Med. Sci., **11**, 163.
8. Spray, G. H. (1956): Proc. Nutr. Soc., **15**, 119.
9. Wohl, M. G. and Goodhart, R. S. (1960): *Modern Nutrition in Health and Disease*. Philadelphia: Lea and Febiger.
10. Olson, O. E., Burris, R. H. and Elvehjem, C. A. (1947): J. Amer. Dietet. Assoc., **23**, 200.
11. Girdwood, R. H. (1959): Brit. Med. Bull., **15**, 14.
12. Harrison, R. J., Booth, C. C. and Mollin, D. L. (1956): Lancet, **1**, 727.
13. Pollycove, M., Apt, L. and Colbert, M. J. (1956): New Eng. J. Med., **255**, 164.

NOTES AND NEWS : BERIGTE

DR. M. M. SUZMAN: 1962 HARVEIAN LECTURER

The Harveian Society of London (founded in 1831 for the advancement of medical science) arranges for the Harveian Lecture to be given once a year.

The Lecturer, as a rule, is selected from among the members of the Society; but occasionally a distinguished member of the medical profession outside the Society is invited to deliver the Lecture.

Dr. M. M. Suzman, of Johannesburg, has been selected to give the next Harveian Lecture on Wednesday, 21 March 1962.

The total membership of the Society is 385. A new Commonwealth Corresponding Membership was recently instituted and Dr. M. M. Suzman was invited to be its first honorary member.

Prof. J. F. Murray of the South African Institute for Medical Research attended the Executive Committee meeting of the Council for International Organizations of Medical Sciences in Paris from 5-7 October. Before returning to South Africa he visited research centres in London, Brussels and Copenhagen.

* * *

MEDICAL LUNCHEON CLUB

ANNUAL BANQUET

The Johannesburg Medical Luncheon Club is holding its 1961 Annual Banquet at the Wanderers Club on Saturday evening, 28 October.

The Chief Guest Speakers are Dr. The Hon. T. E. Dönges, His Excellency, Mr. Joseph C. Satterthwaite (the American Ambassador), His Worship the Mayor of Johannesburg, Councillor Dave Marais and the President of the Southern Transvaal Branch of the Medical Association, Mr. P. Theron.

The Committee of the Club has decided to extend a warm invitation to all members of the medical profession in the Transvaal to apply for table reservations at this Banquet. All inquiries to be made to:

The Honorary Secretary,
401 Medical Arts Building, Jeppe Street,
Johannesburg.

* * *

SOUTH AFRICAN COLLEGE OF PHYSICIANS, SURGEONS AND GYNAECOLOGISTS

An admission ceremony will be held on 3 November 1961 at the Great Hall, the University of the Witwatersrand, Johannesburg at 8.30 p.m.

At the ceremony Prof. I. W. Brebner (Emeritus Professor of Surgery, University of the Witwatersrand) will receive an honorary Fellowship in Surgery, in recognition of his long and meritorious services to the teaching and practice of surgery in South Africa.

Professor Brebner is the first South African medical practitioner to receive this honour.

Tickets for admission to the ceremony may be obtained from:

Dr. P. Knocker, 306 Tafelberg, Esselen Street, Johannesburg. (*Telephone*: 44-7685).

Those wishing to attend the ceremony should obtain tickets beforehand, as most of the seats are reserved.

* * *

NURSES' DAY: 1 NOVEMBER 1961

1 November 1961 will be *Nurses' Day* in South Africa.

The purpose of this *Day*, which is being launched by the South African Nursing Association, is to awaken public attention afresh to the part played by the nurse in the community and to the fact that the term *Nursing* to-day covers a tremendous field.

By courtesy of the churches, the press, radio, screen, many publications and organizations, the thoughts of the people are being drawn to this nation-wide project which is the first of its kind in this country.

Branches of the South African Nursing Association have planned programmes for this *Day* ranging from floats, photographic exhibitions, special meetings and sporting events. One large hospital is holding an *Open Day* on 1 November 1961.

Nursing in South Africa has gone far since in 1658, 6 years after the founding of the settlement at the Cape of Good Hope, a school was started for 'attendants on the sick,' and since the establishment in 1699 of a hospital with a full-time matron—the prototype of the matron to-day.

In 1892 there were 35 registered nurses and 11 midwives in South Africa. In 1957, 20,067 general nurses (male and female, White, Coloured and African) and 13,551 midwives (White, Coloured and African) were registered with the South African Nursing Council, which is the statutory body empowered by Act of Parliament to control nurse registration and education. These figures do not include the mental nurses and nurses for mental defectives who are also registered with the S.A. Nursing Council.

The South African Nursing Association, itself a statutory body, represents the interests of all nurses and is actively concerned, *inter alia*, with the provision of an efficient and adequate nursing and midwifery service and with raising the status and maintaining the integrity of the nursing and midwifery professions.

The opportunities for registered nurses to-day are legion. Following a basic 3-year course undertaken at a hospital nursing school or nursing college (of which there are 134 in South Africa) or a degree course, there are post-basic nursing courses available to befit them for a wide range of posts in the nursing field.

The degree courses at present offered are as follows:

B.A. (Nursing) or B.Sc. (Nursing, University of Pretoria.

B. Soc. Science (Nursing), University of the Orange Free State.

Scholarships and study grants are available for nurses in need of financial assistance in order to enable them to study further in this country or overseas.

One of the newest developments is the decision of the South African Nursing Association to establish a South African College of Nursing for post-basic study. This College, which will be decentralized, will offer courses to nurses of all races.

Internationally as well as nationally, the nurses of this country play an active part in affairs of their profession. As a member country of the International Council of Nurses, South Africa sent a delegation, headed by the President of the S.A. Nursing Association, to the Twelfth Quadrennial Congress of the I.C.N., held in Melbourne, Australia, in April this year.

The nursing profession is strongly aware of its responsibilities to the community. *Nurses' Day* gives its members the opportunity to show how they are meeting these responsibilities and opens the door to better understanding.

* * *

UNIVERSITY OF CAPE TOWN

REFRESHER COURSE FOR GENERAL PRACTITIONERS

22—26 JANUARY, 1962

A Refresher Course for General Practitioners will be held, consisting of lectures, ward rounds and demonstrations in **Medicine, Surgery, Obstetrics** and

Gynaecology and the **Specialties** at appointed times between 8 a.m. and 5.30 p.m. daily form January 22nd to 26th, 1962. Practitioners who wish to spend a further three days to attend special clinics, departments, operations, etc., are welcome to do so but, unfortunately, will not be able to remain in Medical Residence for this extended period as the students will have returned.

The Course will probably include the following:—

A. *Ward Rounds*, with senior members of the teaching staff in Medicine, Surgery, Dermatology, Paediatrics, Obstetrics and Gynaecology.

B. *Panel discussions* by groups of consultants on a variety of subjects including the following:
- Everyday surgical procedures.
- Common surgical lesions in infants and children.
- Acute cardiac disease.
- Hormones in practice.
- Acute trauma—Head, Abdominal and Thoracic.
- Common obstetrical and gynaecological problems.
- Asthma and other allergic disorders.

A feature of these discussions is that ample opportunity will be provided for members of the panel to answer questions raised by the Practitioners attending the Course.

C. *Demonstrations* by the Consultant staff of the following:
- Anaesthetic techniques and minor surgery.
- Minor Gynaecological Procedures.
- Everyday orthopaedic methods.
- Minor medical & paediatric procedures.

D. *A lecture—demonstration* on the following subjects:
- Respiratory infections of childhood.
- Common skin diseases.

E. Sessions *Any questions?*

Special sessions will be provided in Ophthalmology and Otorhinolaryngology, Paediatrics, which a panel of members will answer questions asked by General Practitioners on any subject not covered by the programme of the Course.

The number of practitioners who can be accepted for the Course is restricted.

The fee for the Course will be R10.50, payable in advance to the Registrar, University of Cape Town. *This fee should not be sent until the applicant has been notified that he will be admitted to the Course.*

Board and lodging will be available at Medical Residence for those desiring it (and for their wives) at a charge of R2.10 per day per person.

Applications for admission to the Course, stating whether residential accomodation will be required or not should be submitted as soon as possible to the Registrar, University of Cape Town, Private Bag, Rondebosch, and should reach him before Friday 10 November, 1961.

* * *

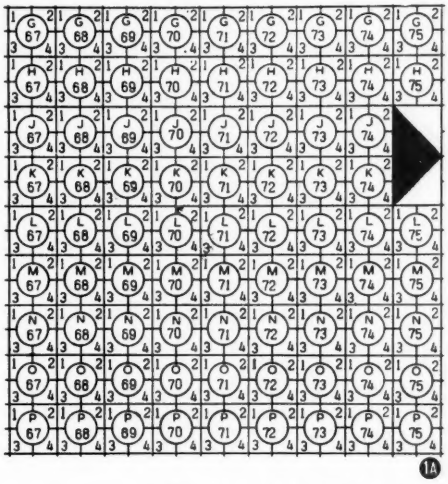
NEW REFERENCE 'FINDER' FOR USE IN MICROSCOPY

The England 'Finder' has been designed to provide an accurate reference system whereby the user of a microscope and slide-mounted specimens can be sure of easily and quickly finding the field of interest. Its simplicity is such that any other viewer (anywhere in the world), using any other microscope and 'Finder', can also be assured of quick location on the same slide.

The system is simple and may be likened to the national grid for map users.

It consists, as Fig. 1A shows, of a glass slide, 3 in. by 1 in., marked with a square grid at 1 mm. intervals. Each square contains a centre bearing a reference letter and number, the remainder of the square being subdivided under 4 segments, numbered 1 to 4. Reference numbers run horizontally (1 to 75) and the letters (A to Z but omitting I), run vertically. The main locating edge is the bottom of the slide, which is used in conjunction with either the left or right vertical edge of the slide, according to the fixed stop of the stage of the microscope, all 3 locating edges being marked with arrow heads.

The label on the 'Finder' should, of course, always appear visually at the bottom left corner when, through most microscopes, the reference image will appear correct.



1A

1B

Fig. 1A. A very much enlarged portion of the England 'Finder' at the side of which is one of the indicating arrows.

Fig. 1B. The point of interest is marked with a cross and will be seen to lie in the third segment of the square of reference K34; thus the 'Finder' reference will be: K34/3.

Use of this England 'Finder' is simple. The specimen slide is marked with a label, on the left, indicating with arrows which sides are to be used for location. The slide is then placed on the stage of the microscope and the bottom long edge is brought into contact with the base stops of the stage and then slid either to the left or right, into contact with the vertical fixed stops, as necessary.

It is essential to obtain the main location of the slide and 'Finder' on the base stops first. After examining the specimen in the normal way and finding a point of interest, it is brought to the centre of the field of view; then, taking care not to alter the position of the fixed stops of the stage, the slide is removed and replaced by the England 'Finder', again bringing the bottom edge in contact first and sliding to the appropriate vertical stop, the label of the 'Finder' being at the bottom left

corner. The reference pattern of the 'Finder' can now be seen through the microscope.

The reference number of the main square is recorded, followed by an oblique stroke and the number of the segment in which the centre of the field of view lies.

The reverse procedure is adopted when re-locating the point of interest, the 'Finder' being placed on the stage, as described above, and the stage adjusted until the appropriate reference square and segment appear in the centre of the field of view.

By removing the 'Finder' and replacing with the specimen slide with the label to the left and the appropriate vertical side in contact with the fixed stop, the point of interest will appear in the centre of the field of view.

Graticules Ltd., the makers of the England 'Finder', have also produced a small self-adhesive label which can be stuck onto the slide-mounted specimen. This label is in effect an enlarged version of one reference square and enables the user to indicate the exact point in the field of view for future reference or for ease of reference for other users. These labels are supplied in sheets of 40 labels per sheet.

Uses for the 'Finder' are legion, in Hospitals, Colleges, Universities, Laboratories, Museums, scientific and nucleonic research, photography; in fact, anywhere where slide-mounted specimens are used with a microscope.

Further details of this 'Finder' and the self-adhesive labels may be obtained from Graticules Ltd., 57–60 Holborn Viaduct, London, E.C.1. England. (Central 2717).

* * *

MEDICAL GRADUATES ASSOCIATION, UNIVERSITY OF WITWATERSRAND

DIARY OF EVENTS: OCTOBER 1961

All Members of the Medical Graduates Association are welcome to attend all Lectures and Ward Rounds listed below.

30 *October* 1961. Rand Medical Discussion Club. (joint meeting with Council of Social Welfare Workers) arranged by Rand Aid, at Shell House, Eloff and Smit Sts. at 8 p.m.

Professor Sheldon, President, World Congress on Geriatrics on *Role of Geriatrics in Social and Medical Work.*

31 *October* 1961. Rand Medical Discussion Club at Harveian Lecture Theatre, Medical School, at 8.15 p.m.

Dr. M. D. Snelling, Deputy Director, Myerstein Institute of Radiology, Middlesex, London, on *Cancer of the Female Genital Tract.*

Institute for the Study of Man in Africa at the Harveian Lecture Theatre, Medical School, at 8.15 p.m.

Clinical Ward Rounds

Mondays: General Hospital. Ward 14-15. Medical. 9 a.m.

Tuesdays: General Hospital. Ward 14. Medical. 9 a.m.

Tuesdays: General Hospital. Ward 22. Surgical. 8 a.m.

Tuesdays: General Hospital. Ward 22. Surgical. 8.30 a.m.

Tuesdays: General Hospital. Ward 12. Medical. 10.30 a.m.

Tuesdays: Princess Nursing Home. Professor Gear Medicine Ward Round. 5 p.m.

Wednesdays: General Hospital. Ward 3-4. Professorial Staff Round. 9 a.m.

Wednesdays: General Hospital. Ward 14-15. Medical. 9 a.m.

Thursdays: General Hospital. Ward 22. Surgical. 8 a.m.

Thursdays: General Hospital. Ward 5. Medical. 10 a.m.

Fridays: General Hospital. Ward 28. Psychiatric. 9.15 a.m.

Fridays: General Hospital. Ward 14-15. Medical. 9 a.m.

Thursdays: Fever Hospital. 8.30 a.m.

Fridays: Edenvale Hospital. Ward 7. European or Ward B, non-European. 12 noon.

Fridays: Coronation Hospital. 2 p.m.

Saturdays: Princess Nursing Home. 2nd Floor. Neurology. 9 a.m.

Tuesdays: Florence Nightingale, 2nd Floor. Mr. D. Fuller. Surgical Cardiological Round. 7.45 to 10 a.m.

Saturdays: Florence Nightingale Nursing Home, 1st Floor. Dr. Fatti. Surgical Cardiological Round, Thoracic Unit. 8.30 to 10 a.m.

Tuesdays: Baragwanath Hospital. Lecture Theatre. Surgery. 1.30 to 2.45 p.m.

Tuesdays: Baragwanath Hospital. Ward 17. Paediatrics. 4 to 5 p.m.

Thursdays: Baragwanath Hospital. Lecture Theatre. Cardiology. 2 to 3 p.m.

Fridays: Baragwanath Hospital. Lecture Theatre. Medicine. 2 to 3 p.m.

Tuesdays: Transvaal Memorial Hospital. 8 to 9.30 a.m.

Weekly Meetings

South African Institute for Medical Research at S.A.I.M.R. Lecture Theatre at 5.10 p.m.

Mondays

23 *October* 1961. *Some Aspects of the Pathology of Idiopathic Heart Disease* by Prof. B. J. P. Becker.

Department of Surgery: *Surgical Forum*: At the Harveian Lecture Theatre at 5.15 p.m.

Tuesdays

24 *October* 1961. *Report on Burns Symposium at Birmingham* by Mr. D. Walker.

31 *October* 1961. *The Use of Radiotherapy in the Treatment of Malignant Disease of the Oral Cavity* by Miss M. D. Snelling.

Inter-Varsity Surgical Forum to be held in Pretoria, on Wednesday, 1 November 1961.

Saturdays. Hospital Lecture Theatre: Vascular Clinic at 8 a.m.

Saturdays. Hospital Lecture Theatre: Surgical Conference at 9.15 a.m.

Annual Golf Tournament. To be held at Glendower Golf Club on Sunday, 22 October 1961. Please telephone 44-7040 (*mornings*).

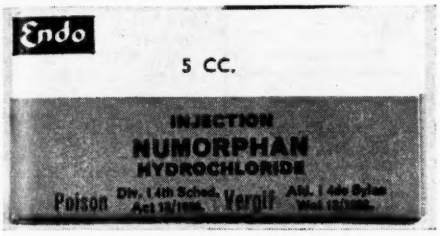
PREPARATIONS AND APPLIANCES

NUMORPHAN

A PARENTERAL NARCOTIC ANALGESIC

Numorphan (oxymorphone) is a recently developed parenteral narcotic analgesic. It will provide pain relief comparable to morphine but with far fewer side effects.

Numorphan provides simple sedation without stupor and will allay the apprehension experienced by the patient being prepared for surgery. Nausea and vomiting are not problems with **Numorphan.** Constipation, respiration and circulatory depression are a rarity.



The pain relief extended by **Numorphan** is usually so satisfactory in quality and duration that fewer repeat doses are required. Fortunately, **Numorphan** dosage may be increased, if necessary, without producing a corresponding increase in side effects.

As an adjunct to anaesthesia **Numorphan** facilitates smooth induction and reduces the amount of anaesthetic agents required.

Indications:
Pre- and Post-operative pain.
As a premedication.
In labour.
In carcinomas.
Dosage: 0.5 to 1.0 cc. every 6 hours.
Route: Intramuscular, intravenous or subcutaneous.
Packing: Multiple dose vial of 5 c.c.
Each 1 c.c. contains 1.5 mg. l-14-hydroxydihydromorphinone hydrochloride.
Further information can be obtained from:
Keatings Pharmaceuticals, P.O. Box 256, Johannesburg.

VELBE

VINBLASTINE SULFATE, LILLY*

Composition: **Velbe** is an alkaloid extracted from *Vinca rosea* Linn., a flowering herb known as the periwinkle. It represents an entirely new class of oncolytic drug and has been found particularly useful in the treatment of Hodgkin's disease. **Velbe** has been used for the palliative treatment of some malignant neoplastic conditions, but there is no evidence that it has in any instance cured human cancer. In susceptible cases, **Velbe** has produced temporary reduction in the size or temporary disappearance of some tumours, relieved pain and other symptoms, and allowed some patients to regain appetite and weight. Such periods of remission have varied from patient to patient.

Velbe has the empirical formula $C_{46}H_{58}O_9N_4$. It is a member of a new class of dimeric alkaloids containing both indole and dihydroindole moieties. The exact structural formula remains to be determined.

The mode of action of **Velbe** is still under investigation. However, Johnson and associates have presented data from tissue culture studies which indicate an antimetabolic action, probably connected with cellular glutamic acid utilization. Palmer *et al* have also demonstrated that treatment of neoplastic cells with **Velbe** *in vitro* has resulted in arrest of their division at the stage of metaphase.

Velbe has desirable activity against susceptible tumours together with a relatively low incidence of thrombocytopenia and side effects.

Use: Although **Velbe** is being used on an experimental basis for the treatment of a wide variety of neoplasms, the drug is not recommended for the treatment of conditions other than those mentioned below until more data have been accumulated. When an accurate diagnosis has been established, a complete evaluation made, and a history (which includes a review of previous therapy) obtained, then the use of **Velbe** may be considered for the treatment of:

1. Generalized Hodgkin's disease.
2. Choriocarcinoma resistant to other available therapy.

Velbe may be used for the treatment of generalized Hodgkin's disease, especially those cases which are unsuitable for local surgical or radiotherapeutic treatment, or for those patients with localized Hodgkin's disease who have failed to respond to local surgical or radiotherapeutic measures.

Dosage: **Velbe** is a potent intravenous drug; dosage must be individualized from patient to patient. It is therefore essential that the dosage schedule outlined in the package literature be *strictly* followed.

Contra-indications: Patients must not receive **Velbe** unless the white blood cell count is at least 4,000 per c. mm. The presence of bacterial infection contraindicates its use. Such infections must be brought under control with antiseptics or antibiotics before the initiation of dosage with this leukopenia-producing drug.

Supply: **Velbe** is supplied in sterile ampoules, each containing 10 mg. of the drug in the form of a lyophilized plug. No. 687, 10 mg., 10c.c. size, rubber-stoppered (Dry Powder), in single ampoules. The ampoules should be stored in a refrigerator to assure extended stability.

*Previously, the generic name was vicaleukoblastine, abbreviated VLB.

HALDRONE

PARAMETHASONE ACETATE, LILLY

Composition: **Haldrone** is a new synthetic and potent corticosteroid with marked anti-inflammatory activity. It is well tolerated, and the incidence of significant untoward reaction is low.

Chemically, **Haldrone** is 6-alpha-fluoro-16-alpha-methylprednisolone 21-acetate.

Uses: **Haldrone** is a potent anti-inflammatory agent. It appears to be about 9 times as potent as hydrocortisone in ACTH suppression tests in man. Also, when croton oil is used to induce irritation of the skin in anti-inflammatory tests and there is concomitant treatment with different doses of several steroids, **Haldrone** seems to be about 10 times as potent as hydrocortisone.

Extensive long-term clinical metabolic studies revealed that sodium retention is unlikely; and, with average dosage, only minimal changes occur in either sodium or potassium excretion. Metabolic studies, in which 6 or 15 mg. of **Haldrone** daily were used, showed that there may be some loss of sodium and chloride with little or no loss of potassium. At a dosage of 6 mg. daily, **Haldrone** may or may not produce a slight increase in urinary excretion of nitrogen and calcium; but at a dosage of 15 mg. daily, urinary excretion of both nitrogen and calcium is likely to increase significantly.

Advantages: **Haldrone** provides predictable anti-inflammatory effect with a minimum of untoward reactions. Gratifying response has been observed in patients transferred from other corticosteroids to **Haldrone.** Psychic effects are minimal; CNS side effects, such as euphoria and restlessness, are not usually encountered. Muscle weakness and cramping do not appear to accompany therapy with **Haldrone,** and only occasionally is there a significant appetite-stimulating effect. Anorexia is almost never seen.

Indications: **Haldrone** has been used successfully in the treatment of:

Collagen Diseases: Rheumatoid arthritis, systemic lupus erythematosus, polyarteritis.

Allergic Diseases: Asthma, hay fever, drug sensitivity.

Dermatological Disorders: Atopic dermatitis, contact dermatitis, poison ivy, neurodermatitis, urticaria, exfoliative dermatitis.

Haematological Diseases: Acquired haemolytic anaemia, idiopathic thrombocytopenic purpura, leukaemia (palliative).

Miscellaneous Disorders: Nephrotic syndrome, gout (initial therapy), ulcerative colitis, adrenogenital syndrome, sarcoidosis, bursitis, pulmonary emphysema, regional ileitis.

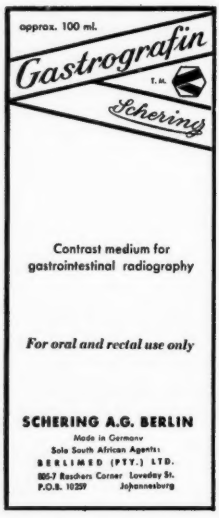
Supplied: Tablets No. 1818, 1 mg., in bottles of 30; Tablets No. 1819, 2 mg., in bottles of 30 and 100.

Remarks: **Haldrone** therapy is seldom accompanied by oedema. Electolyte abnormalities are unlikely. As in the case of other corticosteroids, side effects reported have included rounding of the face, ecchymosis, insomnia, weight gain, hypertrichosis, thinning of the skin, peptic ulcer, occurrence of infections, and increased appetite. In prolonged therapy at suppressive dosage, the features of Cushing's syndrome may occur.

GASTROGRAFIN

Gastrografin is a 76% aqueous solution of the sodium methylglucamine salt of N,N'-diacetyl-3,5-diamino-2,4,6-triiodobenzoic acid with added flavouring agents and a wetting agent. The contrast substance itself is identical with **Urografin** whose use in pyelography and angiography is well established. Because of the additives however, **Gastrografin** is *only suitable for use within the alimentary tract.*

The use of **Gastrografin** does not endanger the patient even in cases of intestinal obstruction or pyloric stenosis, nor when there is a risk of perforation or leakage. No damage is done if **Gastrografin** enters the peritoneal cavity; it creates no problems for the surgeon and moreover can be easily removed by suction.



In cases of partial stenosis the fluidity of **Gastrografin** is of special importance, since it enters the narrowest passages and renders them visible. An additional advantage is that there is no danger, as with barium, that a partial block is made complete as a result of concentration of the contrast medium.

In cases of acute haemorrhage **Gastrografin,** while outlining the mucosal pattern, flows around the blood clot and makes it possible to locate the site of the haemrrohage.

The fact that **Gastrografin** is well tolerated by the tissues and readily absorbed, except by the gastro-intestinal tract, makes it an ideal contrast medium for the visualization of gastro-intestinal fistulae. Even its entry into the tracheo-bronchial tree is not dangerous.

In urgent cases the whole gastro-intestinal tract can be examined radiologically in a very short time, and the site of any obstruction found, without significantly delaying surgery.

In combination with **Gastrografin** barium sulphate loses its tendency to thickening and remaining in the intestine. Combined administration of **Gastrografin** and barium is therefore especially indicated in weak elderly patients or those tending to constipation as well as in cases with diagnosed or suspected megacolon.

Further information from:

Berlimed (Pty.) Ltd., P.O. Box 10259, Johannesburg. *Telephone:* 835-2830.

CORRESPONDENCE

ADVERTISING

To the Editor: You are surely to be congratulated on your excellent editorial on advertising in the issue of 12 August 1961. Some peculiar things are indeed sticking out their heads in our profession. There were the published statements of a worthy in a Provincial Council to the effect that private practice facilities had to be provided at a teaching hospital in order 'to secure the best brains.'

And then we have the introduction of an annual *Akademiese dag* (sounds somewhat like *stryddag*) in the local Civic Centre to which the public is invited. Here lectures are given, demonstrations arranged and photographs taken and published, of the best brains.

Travailler sans souci de gloire
et de fortune.

Cape Town.

MEDICAL COUNCIL BYE-ELECTION

The following statements about the proposed candidates are published for the information of registered medical practitioners.

Ballot papers must be received by the Returning Officer, P.O. Box 205, Pretoria, by 22 November 1961.

Frank, Isidore, M.B., Ch.B. (Univ. Witwatersrand).

Born in Prince Albert, C.P. and brought to Krugersdorp as a young child. Matriculated at Krugersdorp High School and University of Pretoria (then T.U.C.). Spent some time as a primary school teacher, and subsequently enrolled at the newly-established Medical School of the University of the Witwatersrand. After qualification, practised for 8 years in a Transvaal country town, which later served as the *Helfontein* of his book, *A South African Doctor looks Backwards and Forwards.* The difficulties, trials and tribulations of a country doctor are fully recounted, while the question of Poor Whitism is dealt with from a medical and sociological point of view.

After an extensive survey of the condition in this and surrounding areas, he published a clinical study of *Chronic Goitre.*

After relinquishing practice, he attended various clinics and hospitals overseas and resumed private practice in Krugersdorp, where he also became Clinic Medical Officer.

On the outbreak of war, he served for close on 7 years as Medical Officer of Health of Krugersdorp. In 1946 entered the service of the State Health Department and in 1948 was appointed Medical Superintendent of Krugersdorp Hospital. Remained in this post until 1954 when he was appointed Inspector of Hospitals.

At various times was Superintendent in Vereeniging, Coronation and Boksburg-Benoni Hospitals. Appointed to Baragwanath Hospital in 1957.

Freed, Louis Franklin, Hons. B.A., M.A. (Psychology, with distinction) (S.A.), D.Phil. (Sociology) (Pret.), D.Phil. (Social Science) (O.F.S.), M.B., Ch.B. (St. And.), M.D., D.P.M., D.P.H., D.T.M. & H., D.I.H. (Rand), F.R.S.S.Af., F.R.A.I., F.S.S., F.R.G.S.

Member of the New York Academy of Sciences; Part-Time Lecturer on Medical Sociology and Part-Time Lecturer in the Department of Psychiatry, University of Witwatersrand;

First scholar to be appointed an Honorary Visiting Lecturer in the Faculty of Social Science of the University of the Orange Free State, and the series of lectures he gave included *The Principles of Methodology, A Methodological Approach to the Problem of Mental Disorder, The Problem of Crime in the Republic of South Africa: An Integralist Approach, The Principles of Industrial Psychology,* and *The Problem of Prostitution,* etc.

Invited in 1961 to address the Annual Conference of Medical Students of the University of Pretoria on the theme *The Social Sociological Forces within the Human Continum in the Production of the Stress Situation;*

Made a Fellow of the Royal Society of South Africa for his contributions to social psychiatry and social medicine; and a Member of the New York Academy of Sciences for his contributions to the advancement of science;

Invited to serve as South African Editor of *Vita Humana: International Journal for Human Development,* Switzerland and South African Editor of *International Journal of Sexology;*

Played a part in the establishment of the Medical Faculty at the University of Pretoria;

Was Honorary Treasurer of the Actions Committee of the National War Memorial Health Foundation and Chairman of the Johannesburg Branch of the Social Services Association of South Africa;

Was a member of the Council of the Southern Transvaal Branch of the South African Medical Association;

Submitted memoranda to Police Enquiry Commission, Transvaal Provincial Education Commission, Penal Reform Commission, Women's Legal Disability Commission, etc.;

Was invited to address an Annual Conference of the Dutch Reformed Church, held in 1947 at the University of Witwatersrand on *Social Evils,* and also a group of Ministers of the same Church on *Sex Education;*

Evolved the concept of the *human continum* in the philosophy of medicine; and in reference thereto the citation published in 1959 by the University of the Orange Free State stated *inter alia*:

> 'Aan hom is te danke die ontwikkeling van die begrip, *the human continum* in die filosophie van die mediese wetenskap asook van die metodologiese beginsels met betrekking tot mediese, medies-sosiale en medies-sosiologiese vraagstukke . . .'.

His biography is published in *Who's Who in British Science, The International Who's Who,* and, *Who's Who in World Jewry.*

Publications include:

1. *The Problem of European Prostitution in Johannesburg: A Sociological Survey* (1949) (Prescribed for Social Science students at the Universities of Pretoria, Witwatersrand and South Africa).
2. *Sex Education in Transvaal Schools* (1937).
3. *The Social Aspect of Venereal Disease* (1951).
4. *The Philosophy of Sociological Medicine* (1948).
5. *The Need for a Division of Criminal Science in the Department of Justice of the Union Government* (1948).
6. *Crime,* a Chapter in *Social Medicine,* Ed. by Cluver (1951).
7. *Findings of an Investigation into a Group of Patients Presenting with the Symptoms of Schizophrenia* (1953).
8. *A Methodological Approach to the Problem of Mental Disorder* (1956).
9. *Medico-Sociological data in the Problem of Homosexuality.*
10. *The Problem of Depression in Psychiatry* (1957).
11. *The Psycho-Sociology of Neoplasia* (1958).
12. *An Enquiry into the Causality of Cancer: A Functionalist Approach* (1958). (In conjunction with G. Giannopoulos).
13. *The Use of Methodological Principles in the Investigation of a Case of Trichomoniasis* (1956).
14. *The Problem of Crime in the Union of South Africa: An Integralist Approach* (1959), etc.
15. *The Social Aspect of Alcoholism: An Integralist Approach* (1960), etc.

le Roux, J. J. du Pré, L.R.C.P. & S. (Edin.), L.R.F.P.S. (Glasg.), D.P.H. (Univ. Witwatersrand).

Jonathan Johan du Pré le Roux was born at Ceres, Cape Province, on 3 August 1900. He was educated at the Boys' High School, Paarl, and studied first at the University of Cape Town and then at the University of Edinburgh, where he qualified in medicine in 1923. During 1923 and 1924 he was in private practice in England. He returned to South Africa in 1924 and, after further private practice at Hermanus, Cape, and elsewhere, he joined the Government service in 1927 as medical officer at the Westfort Leprosy Institution, Pretoria, of which, after obtaining the D.P.H. at the University of the Witwatersrand, he became Medical Superintendent in 1933.

Dr. le Roux left the Government service in 1936 on appointment as Medical Officer of Health of the Municipality of Boksburg, Transvaal, in which position he served for 3 years before rejoining the Union Health Department in 1939, where he was Assistant Health Officer at Cape Town and then Deputy Chief Health Officer at East London and Pretoria. On 1 March 1952 he was appointed head of the Department as Secretary for Health and Chief Health Officer. He retired from this position on 3 August 1960 on attaining pensionable age.

As Secretary for Health he was automatically a member of the Medical Council. He is now offering himself for service on the Council as an elected member.

Schneider, Tobias (Teddy), M.D. (Rand), F.R.C.P. (Edin.).

Graduated M.B., B.Ch. (Rand), 1927; M.R.C.P. (Eng.), 1940; M.D. (Rand), F.R.C.P. (Edin.), 1958.

Bronze Medal, Medical Association of South Africa, for meritorious service.

In general practice 1930-45.

Specialist Physician, Johannesburg, 1945 to the present.

Posts Held at Present:

Physician and Physician-in-Charge, Diabetic Clinic, Johannesburg General Hospital and University of the Witwatersrand.

Chairman, Ethical Committee of Federal Council, M.A.S.A.

Member of Federal Council, M.A.S.A. from 1946 to the present.

President, Association of Physicians of South Africa.

Chairman, Metabolic, Endocrine & Diabetic Society of South Africa.

Chairman, Press Liaison Committee, Southern Transvaal Branch, M.A.S.A.

Member of Branch Council, Southern Transvaal Branch, M.A.S.A.

Member of Medical Executive Committee, College of Physicians, Surgeons & Gynaecologists of South Africa.

Positions Previously Held:

President, Southern Transvaal Branch, M.A.S.A.

Chairman, Southern Transvaal Branch, Association of Physicians of South Africa.

Honorary Secretary, Southern Transvaal Branch, M.A.S.A.

President, Medical Graduates Association, University of the Witwatersrand.

Member of Steering Committee, College of Physicians, Surgeons & Gynaecologists of South Africa.

Captain, 25th: Field Ambulance, S.A.M.C.

Chairman, General Practitioners War Fund.

Turton, Edwin Wilberforce, M.B., Ch.B., (Univ. Cape Town).

Dr. Edwin Wilberforce Turton was educated at Grey College, Bloemfontein, where he had a distinguished scholastic and athletic career. A testimony to his popularity was that he was School Captain for 2 years consecutively. He represented his school in the first teams at rugby, cricket, tennis, water polo and boxing (light heavy-weight).

He spent his first 3 academic years at the University of the Witwatersrand and the last 3 at the University of Cape Town, graduating to the degrees of M.B., Ch.B. in 1939. Whilst at Cape Town, he played in the University first teams in rugby, cricket and tennis.

In 1940 Dr. Turton settled in Boksburg and was appointed part-time general practitioner registrar at the Boksburg-Benoni Hospital until 1945, when he was appointed part-time general practitioner surgeon at the same hospital. He was a member of the Boksburg-Benoni Hospital Board from 1944 to 1959 and served as Chairman of the Board from 1953 to 1959. His yeoman service was terminated in that year by the introduction of the Hospitals Ordinance of 1959 of the Transvaal Province. In 1958 he was appointed a member of the Planning Committee of the Transvaal Provincial Department.

Besides carrying on a busy general practice in Boksburg, Dr. Turton has served on the School Board of the Voortrekker School, Boksburg from 1948 to 1960. He was Chairman of the Jubilee Home, which is administered by the Department of Social Welfare, from 1946 to 1956.

Dr. Turton's devoted service to the Medical Association commenced in 1940 when he was elected as a member for the East Rand Division of the Southern Transvaal Branch of the Association. With the formation of the Eastern Transvaal Branch of the Association, Dr. Turton was elected the first President of the Branch. He has been a member of Federal Council since 1947 to the present day, with a short break from 1952 to 1954. In 1951 he was elected to the Executive Committee of Federal Council and served in this capacity for the year 1951-52 and again from 1954 to the present day. Federal Council honoured him by electing him Vice-Chairman of the Council in 1957 and Chairman of Council in 1960.

He has been a member of the Transvaal Augmented Executive Committee since 1947. In 1946 he served on the Mediation Committee of the Association. These negotiations were successfully concluded with the Transvaal Provincial Administration.

Dr. Turton, in addition to his other activities, is at present part-time Senior Medical Officer Administrative of the Railway Sick Fund. He has been Chairman of the Mines Medical Officers' Group and the Railway Medical Officers' Group of the Association and was also a member of the Steering Committee of the General Practitioners' Group of the Association.

The Eastern Transvaal Branch recommended to Council that Dr. Turton be awarded the Association's Bronze Medal for meritorious service.

C.T. LTD.